Teratological Evidence as to the Heredity of Acquired Conditions.

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THE subject of congenital malformations is one which, strangely enough, has met with but little notice amidst the mass of evidence which has been brought forward, on one side or the other, as to the transmissibility of acquired characters, a question which has provoked, and is provoking, so much controversy.

Yet it is certain that the consideration of the question from its teratological aspect ought to be of some service, since, in the first place, there are two groups undoubtedly amongst congenital malformations, blastogenic and somatogenic, to use Weismann's terms, though it may not always be easy to assign a given defect with certainty to either. And, again, it ought to be possible to throw some light upon the difficult question of the origin, development, and fate of a variation, from the copious mass of literature which exists in relation to teratological subjects. It seems probable that the reason why this field has been so little explored in the present connection, is that the etiological side of teratology has, up to the present, been so much neglected. Professor Cleland, who has done so much for the study of the subject in this country, has very aptly remarked that teratology should be defined as "pathological embryology." Before, however, much practical information can be gathered from the subject, it is, above all things, necessary that its classification should be arranged upon an etiological basis. The author just quoted remarks in an essay on "Teratology, Speculative and Causal, and the Classification of Abnormalities"* :—"If the advantage which biological doctrine has hitherto derived from teratology has not been great, the reason has lain, not in the barrenness of the teratological field, but in the small amount of progress made in determining the true nature of teratological phenomena. Even after the old conception of *lusus naturæ* had been thrown aside, the most imperfect notions continued to prevail

* 'Memoirs and Memoranda in Anatomy,' vol. i. 1889.

both with regard to abnormalities by excess and many of the more important abnormalities by defect. But we have now arrived at a time when no thoughtful enquirer can be longer content with merely cataloguing deviations and bestowing upon them sesquipedalian names such as were perhaps justifiable in the days of the elder St.-Hilaire. Investigation has entered upon a more strictly causal stage; and, to my thinking, it becomes evident that teratology has an important work before it in relation to biological science generally, by demonstrating the presence of potentialities which in the normal organism lie dormant, but nevertheless must exist, or they could not in exceptional circumstances show their presence."

In the present essay my intention is to examine the recorded groups of hereditary malformations, with a view to ascertain their nature, so far as may be possible, whether blastogenic or somatogenic. In order to do so, it will be necessary, however, for me to preface this portion of my paper with a short account of the groups into which malformations should be causally arranged. At the conclusion of the main part of the paper, that, namely, which deals with hereditary malformations, I shall add a few remarks on certain points in connection with the causation of malformations which have not, I think, up to the present received sufficient attention.

I may perhaps here state that this paper was not undertaken with a view either to support or to oppose Weismann's views. My desire has been to examine the subject from a teratological standpoint and to record the result of my observations.

Section I.—Varieties of Malformations.

As has been already suggested, any etiological classification of anomalies at the present time must be more or less tentative; yet I believe we may reasonably strike a line of division between two great groups, each of which may be again subdivided. The first group consists of malformations which are due to some error in the amount of formative material, and the second of those which are not. The former group may be spoken of as non-mechanical, and the latter as mechanical, provided it be understood that these terms are not used in the sense in which they are applied to the ordinary occurrences of development by His, Weismann, Roux, and other writers. Errors of material may be in two directions,

viz. excess or defect. To the former class belong double monstrosities, complete and incomplete, unilateral or partial hypertrophies, and excesses such as polydactyly (at least the majority of cases), polymastia, with others, often not recognized unless a dissection happens to be made, such as accessory carpal bones, splenculi, &c. Having discussed the sources of this excess of material in a former paper *, I shall not delay here further on that point. To use Weismann's term, malformations of this class are, with probably very few exceptions, inherent in the germ or blastogenic. Deficiency of material leads to defect of size or parts. Thus, to this group belong dwarfs and some cases of ectromelia, ectrodactyly, microphthalmus, and anophthalmus, with possibly most of the non-mechanically caused clefts, such as cleft-palate, persistent branchial cleft, and coloboma oculi. How is this deficiency of material caused? I believe in at least two ways:—(1) By faulty segmentation during the extrusion of the polar bodies, whereby too much formative material is got rid of. (2) By the very early destruction by some morbid process of early segmentation-spheres. Roux's experiments, which I have quoted in my paper already mentioned, and an account of which will also be found in a recent address by Mr. Poulton †, show that even the first few lines of segmentation cut off morphological areas the destruction of any one of which would lead to the non-development of the part which it was destined to form. It is, of course, possible that to the two preceding causes should be added that of an original paucity of material in ovum or spermatozoon prior to the extrusion of the polar bodies. The first and last causes would be inherent in the germ, and therefore blastogenic; but what of the second? Early though it occurs, it can scarcely be looked upon as other than somatogenic in its nature. The effects, however, may not be distinguishable from those due to the blastogenic causes mentioned. Here, of course, the difficulty of discrimination renders any inferences difficult and dangerous. The second great group of mechanical malformations is obviously entirely somatogenic in its nature; and the only dubious point connected with it is the difficulty of deter-

* "The Origin of Double Monstrosity," Journ. of Anat. and Phys. vol. xxiii. p. 390.

† "Theories of Heredity." An Address, reprinted from the 'Midland Naturalist,' Nov. 1889.

mining in all cases whether a given defect belongs to it or to one of the other divisions. This group may be divided into the following classes:—(1) Clefts of various kinds, due to the presence of tumours or to fluids consequent upon inflammation. Certain forms of cleft-palate belong to the former category, abdominal and spina clefts to the latter. (2) Deficiencies of extremities &c. due to inflammation of tissues leading to formation of fibrous tissue, its contraction, and the strangulation of growing parts. As examples of this may be named certain forms of peromelia and (?) atresia ani. (3) Deficiencies due to amniotic pressure. (4) Deficiencies due to adhesions of the amnion &c., including the so-called intra-uterine amputations. Having thus very briefly sketched the main lines of classification, it will be necessary in the next section, as each malformation comes up for examination, to consider at greater length and in more detail its special etiology.

Section II.—Nature of Hereditary Malformations.

In this section I purpose examining those malformations whose hereditary nature is established, *seriatim*. In connection with the etiology of each will be considered its nature, whether blastogenic or somatogenic. I shall have to draw largely from a former paper of my own "On Congenital Malformations and Heredity" *, in which are collected a number of recorded cases of such malformations, but without reference to their etiology. It will be convenient in the subsequent part of this paper to group the malformations under the classes which have been indicated in the first section.

Part 1.—*Malformations by Excess.*

The most important members of this group, all the forms, that is, of double monstrosity, have to be entirely excluded from the present inquiry. An enormous majority are non-viable, or only maintain a separate existence for a few hours or days; and the remainder are, even if married, sterile. The same remark applies, as will appear later, to most of the major and even to some of the minor forms of abnormality. We can, however, with profit examine two classes of cases not commonly grouped under the

* Proc. Birm. Phil. Soc. vol. vi. pt. 1.

head of malformations, and yet closely allied to Double Monsters, viz. Giants and Homologous Twins. According to the views expressed in the first section, a little more formative material plus equivalent division, or even the latter alone, would transform the giant into a double monster, or the latter into a pair of homologous twins. A few notes, then, upon these two groups are quite pertinent to the subject in hand.

Giants.—These are generally sterile, according to the best authorities. I. G. St.-Hilaire says of them *:—"Ils sont ordinairement impuissants, et sont très promptement énervés par les plaisirs de l'amour. Le défaut d'aptitude des géants aux fonctions génératrices ne saurait étonner chez des êtres épuisés et affaiblis par la rapidité et l'excès de leur accroissement." And in a footnote he adds, "chez quelques géants, l'érection est même presque complètement impossible." Topinard † agrees with St.-Hilaire on this point. Again, gigantic children are generally still-born when the mother is of the ordinary size. Thus, Dr. Parvin ‡ states:—"Some women, though their labours are at the normal period, give birth to children whose great development presents a more or less serious obstacle to labour, and still-births are not unfrequent." Kormann refers to a case in which the child presenting by the breech was still-born, and weighed 9·8 kilos. (normal weight 3–4 kilos.).

With regard to the frequency of occurrence of large fœtuses, Jaggard says §:—"Variations in weight at term between six and nine pounds are by no means rare—an infant over nine pounds is not common; while heavier weights are progressively rarer. Out of 1000 infants, Dr. Parvin saw but one that weighed eleven pounds (Parvin's Obstet. p. 138). Of 1156 infants born in the Maternity Hospital, the heaviest weighed 12 pounds." Giants thus resemble double monsters in rarity, non-viability, and sterility. Instances are, however, on record where giants have borne gigantic children. Mrs. Bates was 7 ft. 9 in. in height, and her husband 7 ft. 7 in. She bore him an infant which weighed $23\frac{3}{4}$ pounds (normal av. $7\frac{1}{3}$ pounds); its height was 30 inches

* 'Anomalies de l'Organisation,' vol. i. p. 183.

† 'Eléments d'Anthropologie générale,' p. 436.

‡ 'American System of Gynecology and Obstetrics,' vol. i. p. 753.

§ 'American System,' vol. i. p. 214.

(normal 20–21), breast measure 27 in., head 19 in., foot $5\frac{1}{2}$ in. The liquor amnii amounted to 5 gallons, and the secundines weighed 10 pounds. Again, the Nova-Scotia giantess, according to Harris *, bore a child which weighed $28\frac{3}{4}$ pounds. Thus giantism, if the word may be coined, is, or may be, hereditary. Moreover, as St.-Hilaire remarks, the brothers and sisters of giants are generally of large stature; they come, in fact, of tall families, to quote the popular phrase, embodying the popular belief, correct in this case, of the heredity of stature. Is this variation blastogenic or somatogenic? I have no hesitation in assigning it to the former class. If it be urged that the condition is due to the superior nourishment of the child by the mother whilst pregnant, and is therefore blastogenic, I would reply (1) that gigantic children are not necessarily the product of well-nourished mothers; for, were it so, by this time our Royal and some of our noble families ought to be like children of Anak; and (2) that giants are at birth not always larger than ordinary children. They possess the potentiality of great size, a potentiality which is, I believe, inherent in the germ Cohnheim puts this point of the potentialities of growth so well, that I shall quote his own words †:—"Der Neugeborene bringt nicht die Geschwulst, sondern lediglich das überschüssige Zellmaterial mit auf die Welt, aus dem unter günstigen Verhältnissen später einer Geschwulst herauswachsen kann. Nur möchte ich noch einmal nachdrücklich bitten, das Sie Sich nicht zu sehr an dem Wortlaut des 'überschüssigen Zellmaterials' binden wollen; vielleicht wäre es selbst richtiger, statt dessen von demjenigen Material zu sprechen, welchem die Potenz zu späterer Geschwulstbildung beiwohnt. Denn auf diese Potenz kommt es an, die übrigens keine andere Eigenschaft ist, als wie so ungemein häufig bei der individuellen Vererbung und Entwicklung sich geltend macht. Wenn bei dem Sohn eines langnasigen Vaters die bis dahin völlig proportionirte und ganz unauffällige Nase im 8 oder 10 Lebensjahr oder noch später grosse Dimensionen annimmt und ungewöhnlich lang wird, so zweifelt Niemand daran, dass die Potenz zu diesem Wachsthum von Anfang an in der Nase existirt hat—obschon die sorgfältigste und genaueste mikroskopische Untersuchung in den ersten

* New York Med. Record.

† 'Allgemeine Pathologie,' Bd. i. S. 740.

Lebensjahren ausser Stande gewesen sein würde, jene Potenz aufzudecken."

In the same way the child-giant brings into the world with him the potentialities of his stature and size. On the other hand, it may be urged that post-natal nourishment may be the factor; and Bishop Berkeley's giant* may be cited in confirmation. To this it may be replied, that post-natal nourishment can affect the height only within very narrow limits; for otherwise there would be none but dwarfs in those parts of Ireland, for example, where potatoes and seaweed are the staple foods, and none but giants amongst the classes whose circumstances have been easy for generations past. And, as far as M'Grath is concerned, the instance is an isolated one; and there is no evidence to prove what the unfortunate man's stature might have been had he not been subjected to the episcopal tender mercies. On the whole, then, I think it may be said that giantism is an occasionally hereditary condition, blastogenic in its nature.

Homologous Twins.—It is an unfortunate circumstance that the confusion which has existed with regard to true or homologous twins and the other form, vitiates all the statistics as to twins, so far as I have examined them. True twins, which are always of the same sex, enclosed in the same membranes, generally strikingly like one another, and the product of one ovum, are of course totally different from twins the product of two ova, enclosed in separate membranes, not necessarily of the same sex, nor more alike than children of the same family are, or may be. This fact has, however, not been taken into account by those who are responsible for the statistics of twins; and it is consequently impossible to say whether the facts observed relate to both classes or only to one. Subject to this reservation, it may be said that the bearing of twins is certainly hereditary—"runs in a family," as the phrase has it. This is also true of multiple births beyond the number of two. Thus Osiander† gives a case where a woman, herself a twin, was the mother of 38 children, and died in childbed after delivery of twins. One of her daughters, who was born with three others at a birth, had 32 children at 11 confinements. The following statistics given

* 'Philosophical Survey of Ireland,' London, 1777, p. 187.

† 'Handb. d. Entbindungskunst,' 1 Th. 1 Abth. S. 319. Quoted in Edinb. Med. Journ. vol. iii. p. 1143.

by Gœhlert * are of interest in this connection. He first gives a table, from amongst the cases which he has collected, of the pairs and sexes :—

	No.	Male.	Female.	Per cent.
Similar. Two males	61	122	....	29·76
„ females ..	52	....	104	25·36
Dissimilar	92	92	92	44·88
Total	205	214	196	100·00

In examining this table it must, of course, be remembered that the first two lines need not necessarily relate solely to homologous twins. It is quite as possible for two children, of the same sex, yet not true twins, to be born at a birth, as it is for two of different sexes. Passing to the subject of heredity in twinning, Gœhlert states that there is a direct heredity from twin to twin as well as an indirect. That this is not more observable is, he remarks, due to the fact that so many twins die in childhood, only seldom reaching maturity, and, of course, then not always bearing children. He believes that in 132 of 192 cases selected from Royal pedigrees which he carefully examined, the influence of heredity was to be observed. The figures are given in the subjoined table :

	From Father.	From Mother.	Pairs.
Direct inheritance	5	11	16
Indirect „	57	55	112
Direct and indirect	..	..	4
	62	66	132

All that can be said is, that it is at least highly probable that the production of true twins is hereditary.

I have stated that twins are not usually regarded as abnormalities; yet the two are much more closely related than is generally thought. Dr. Mitchell † shows this clearly in a paper, the conclusions drawn in which are :—(1) Among imbeciles and idiots a much larger proportion is actually found to be twin-born than among the general community. (2) Among the relatives of imbeciles and

* "Die Zwillinge," Virchow's Archiv, Bd. 76, S. 457.

† Med. Times & Gaz. Nov. 15, 1862.

idiots twinning is also found to be very frequent. (3) In families, when twinning is frequent, bodily deformities (of defect and excess) likewise occur with frequency." This last conclusion points in the direction of the blastogenic nature of twinning.

Those who hold the opinion that the mother's state of nutrition may be a potent factor, may derive some support from Dr. Matthews Duncan's conclusion, drawn from numerous statistics *, that "the largest number of twins is produced by women of from twenty-five to twenty-nine years of age †; and on each side of this climax of fertility in twins there is a gradually increasing falling-off in their number as age diminishes on the one side, and increases on the other." That nutrition has at least a large influence in the determination of sex, is a proposition supported by Geddes and Thomson ‡; and, as they appropriately ask, and the question might equally apply to the subject of twins, were nutrition proved to be a factor in its production, "How does Weismann explain the determination of sex, which illustrates an outside influence penetrating to the reproductive cells?" It may just be noted, in connection with this subject, and with the last-named authors' view that superior nutrition tends to the production of the anabolic female sex, that the larger number of twins belong to that sex. To sum up this subject, it must unfortunately be said that beyond the statement that twinning is hereditary, it is impossible, in the present chaotic state of statistics on the subject, to draw any definite conclusions in favour of either side of the argument.

Having disposed of these points, I may now proceed to the other malformations partaking of the nature of excess.

Polydactyly.—This is one of the most strongly hereditary forms of malformation. Broca § and M°Kellar ‖ have both recorded cases where it has passed through six generations, and Muir ¶ one through five. One of the most completely recorded cases is that by Clement Lucas, which is here reproduced (p. 457) **.

* 'Fecundity, Fertility, and Sterility' (Edinb. 1871), p. 100.

† Nutrition being probably at its best during those years.

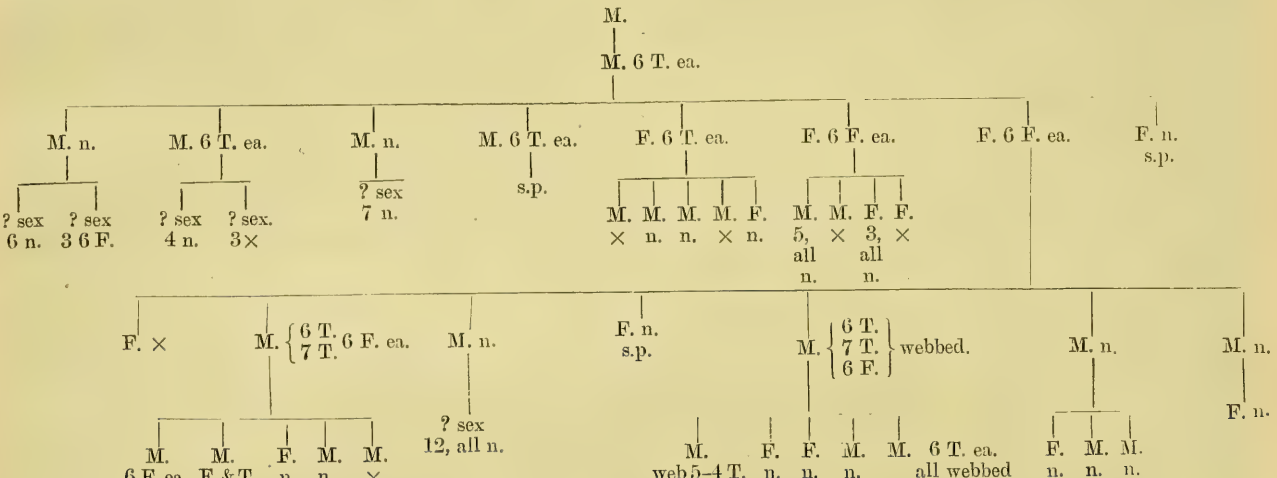
‡ 'The Evolution of Sex' (London, 1889), p. 53.

§ Acad. de Méd. Paris, quoted in Lond. Med. Rec. vol. vi. p. 91.

‖ Glasgow Med. Journ. May 1870.

¶ Ib. vol. xxi. p. 420.

** Guy's Hospital Reps. vol. xxv. Abbreviations: T.=toes; F.=fingers ea.=both members affected; s.p.=sine prole; ×=affected.


M.
M. 6 T. ea.
M. n.
? sex
6 n.
? sex
3 6 F.
M. 6 T. ea.
? sex
4 n.
? sex.
3×
M. n.
? sex
7 n.
M. 6 T. ea.
s.p.
F. 6 T. ea.
M. M. M. M. F.
× n. n. × n.
F. 6 F. ea.
M. M. F. F.
5, × 3, ×
all all
n. n.
F. 6 F. ea.
F. n.
s.p.
F. ×
M. { 6 T. 7 T. } 6 F. ea.
M. n.
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12, all n.
F. n.
s.p.
M. { 6 T. 7 T. 6 F. } webbed.
M. n.
M. n.
F. n.
M. M. F. M. M.
6 F. ea. F. & T. n. n. ×
M. F. F. M. M. 6 T. ea.
web 5–4 T. n. n. n. all webbed
6 F. ea.
F. M. M.
n. n. n.

It is no exaggeration to say that scores of minor cases might be cited were there any advantage in doing so *. It is more important to consider the etiology of the affection. The different forms may be classified in various ways: Gaillard †, for example, dividing by the amount of excess present, a somewhat important factor, as will appear. Thus: "1st. The finger is only bifid, articulating with the metacarpus by a single head. 2nd. Two fingers are placed in juxtaposition, being articulated side by side, only one articular capsule surrounding the head of the metacarpal bone. 3rd. Rarest: the supernumerary finger is entirely isolated, and implanted on a special articular surface of the metacarpus, the superfluous part being almost always a thumb or great toe." Or, again, superfluous fingers may be classified into marginal and central—those, that is, connected with the edge of the thumb or little finger, and those connected with some other finger. That these are different in their nature has been recently pointed out by Kollmann ‡. He says:—"Meine Rudimenttheorie nur den ulnaren und radialen Strahl (Vordaumen" [this had been previously described as præpollex by Bardeleben] "und doppelten kleinen Finger) als Hyperdaktylie verständlich machen soll; andere Arten der Hyperdaktylie gehören bis auf weiteres in das Gebiet der Teratologie. Um Missverständnissen vorzubeugen, wiederhole ich deshalb; Es giebt keine Stapedifera mit mehr als fünf Fingern, aber solche mit fünf Fingern und mit Spuren eines ulnaren und radialen Strahles (Mensch, viele Säuger, Reptilien und Batrachier). Diese Spuren liegen oft als schwer erkennbare Rudimente unter der Haut. In Fällen von Hyperdaktylie des Menschen vergrössern sich diese Rudimente und treten verschieden entwickelt aus der Haut hervor. Der Rückschlag aus diesen Rudimenten liefert wahrscheinlich stets nur Rudimente, d. i. verkümmerte Finger. Hyperdaktylie ist keine pathologische, sondern eine theromorphe § Erscheinung und weist auf eine Reduktion von Strahlen hin, welche bei der Umformung der

* For a very full list, see a paper by Dr. Fackenheim, 'Jenaische Zeitschr. für Naturwissenschaft,' Bd. xxii. N. F. xv. S. 343.

† Gaz. Méd. 1862, No. 43. Quoted by Annandale, 'Diseases of the Fingers and Toes' (Edinb. 1865), p. 26.

‡ 'Mitth. aus d. Anat. Inst. im Vesalianum zu Basel,' 1889.

§ "Θήρ-θήρος, das Thier, auch Thiermensch, φῆρ woron das lat. ferus, ferox, &c."

Fischflosse in eine Batrachierhand mit aufgenommen wurden. Hyperdaktylie des Menschen is demnach eine besondere Form des Atavismus."

As to the mesial form, there is more difficulty in deciding. According to some authorities, as, for example, Ahlfeld*, polydactyly is the result of the compression of the growing digits by amniotic bands, the depth and amount of the excess depending upon the extent of the constriction. Two points should, however, be noted in this connection:—(1) The malformations which are accounted for by pressure of the membranes or by constriction from bands derived from them, or from other sources which will be more particularly considered when the subject of peromelia is dealt with, are gradually being reduced in number as our knowledge increases. Many conditions attributed to these causes are now satisfactorily accounted for in other ways. In connection with the present subject, it may be said that there is little, if any, evidence to show that amniotic bands have ever anything to do with the production of polydactyly. (2) It is not easy to understand how a malformation caused in this way could become hereditary. The heredity, if any exist, must run in one of two lines—an heredity of the amniotic bands causing the malformation, or an heredity of a malformation first caused by a band. The first, which, by the way, has been advanced, seems too improbable to require much consideration. The coincidence by which an inflammatory band could form in exactly the same position, and produce the same effect in the pregnancies of several generations, is one which can scarcely be admitted. And the second mode would be nothing more or less than the inheritance of a mutilation. But Weismann has proved, I think it will be universally acknowledged, in his various writings, that a mutilation has at least never been shown to have been transmitted. Laying aside, however, these two considerations, we have two kinds of polydactyly—the marginal, which it is highly probable is always atavistic and blastogenic; and the central, which may be due to excess of material in the germ, and therefore blastogenic, or, possibly, to amniotic bands, and therefore somatogenic.

It is instructive, then, to inquire which of these is the most common. According to Förster, a supernumerary little finger

* 'Die Missbildungen des Menschen' (Jena, 1865), S. 43.

is the most common form, a thumb or great toe comes next in order, least commonly is the digit one of the central group. I have no data to decide whether the same rules apply to the hereditary cases; but I have little doubt that it would be found so. In the celebrated case quoted by Devay *, where the hereditary malformation was more widely spread within a circumscribed area than in any other with which I am acquainted, the supernumerary digit appears to have been a thumb. I think that this case is worth describing in detail. "Il s'agit d'une véritable endémie de *sexdigitisme*, d'une population entière qui .... a été frappée de cette bizarre anomalie. Nous devons la connaissance de ce fait singulier à notre savant confrère, le docteur A. Potton, qui l'a observé sur les lieux mêmes. Il existe dans le département de l'Isère, non loin de la Côte-Saint-André et de Rives, un tout petit village nommé Izeaux, isolé, perdu en quelque sorte autrefois, au milieu d'une plaine, si non complètement inculte, du moins très-pauvre, dite la plaine de Bièvre. Les chemins, les communications dans ce pays peu fertile, étaient difficiles, si non impraticables. Les habitants d'Izeaux, simples, presque abandonnés à eux-mêmes, n'entretenaient que des rapports éloignés avec les populations environnantes, sans se mélanger avec elles; ils se mariaient constamment entre eux, et ainsi fréquemment en famille. A la fin du siècle dernier, de cette manière de faire, de ces alliances constantes entre parents était née et entretenue par elle une monstruosité singulière, qui, il y a trente-cinq à quarante ans, frappait encore presque toute la population. Dans cette commune, hommes et femmes étaient porteurs d'un sixième doigt, d'un doigt supplémentaire implanté aux pieds et aux mains. 'Lorsqu'en 1829 et en 1836,' dit M. Potton, 'j'ai observé ce bizarre phénomène, déjà, chez quelques sujets, il n'existait qu'à un état plus ou moins rudimentaire; chez plusieurs, ce n'était qu'un gros tubercule, au centre duquel cependant on rencontrait un corps dur, osseux; l'apparence d'un ongle plus ou moins formé terminait cet appendice, fixé latéralement en dehors, *à la base du pouce*†. La personne qui m'accompagnait, bien qu'étrangère à la médecine, me faisait observer qu'une heureuse transformation tendait à s'opérer, que de notables changements dans cette défectuosité organique s'étaient établis depuis que les habitudes de la population s'étaient modifiées par la force des

* 'Du Dangers des Mariages Consanguins' (Paris, 1862), p. 95.

† Italics mine.

choses, par le progrès, depuis que, les voies de communications étant devenues meilleures, les rélations à l'extérieur plus fréquentes, les alliances se contractaient dans des conditions plus favorables; depuis, en un mot, que le croisement des races avait lieu. En 1847 j'ai eu occasion de voir un chef d'atelier, originaire de cet localité, fixé et marié à Lyon. Il était porteur du vice de conformation signalé; il était père de quatre enfants, qui n'avaient point le stigmate paternale. A l'heure qu'il est, d'après les renseignements circonstanciés pris auprès de médecins de la localité, cette anomalie pathologique a presque complètement disparu.'"

To sum up, then, the subject of polydactyly, it may at least be said that there is no proof that any of the hereditary cases are somatogenic. I should go further myself, and say that there is every reason to believe that they are blastogenic.

Supratrochlear Process.—Struthers * has recorded a case in which this curious process or hook was hereditary. It is atavistic and undoubtedly blastogenic in its nature.

Polymastia.— Accessory breasts may exist on the thigh, groin, vulva, back, or other parts of the body, the following table giving Leichtenstern's † figures from an examination of 105 cases :—

The accessory gland was		on the anterior surface of the thorax	in	96.	
„	„	in the axilla	„	„	5.
„	„	on the back	„	„	2.
„	„	on the acromion	„	„	1.
„	„	on the thigh	„	„	1.

The condition may be hereditary, as in the case narrated by Robert‡ of a woman who had a third breast on the exterior of the left thigh placed 4 inches below the great trochanter, with which she had suckled several children; her mother had a third thoracic breast. Woodman § records a case where a woman and her child were both possessed of three thoracic mammæ. As to the cause of the malformation, one would not think that there need be much difference of opinion. Ahlfeld ‖, however,

* Lancet, Feb. 15, 1873.

† Virchow's Archiv, Bd. 73, S. 222.

‡ Journal de Physiologie, tome vii. no. 2.

§ Trans. Obst. Soc. vol. ix. p. 50.

‖ 'Die Missbild. d. Menschen' (Leipzig, 1880), S. 110.

has committed himself to the statement that they are due to the action of the amnion. "Am wahrscheinlichsten ist mir die Möglichkeit, dass durch den Druck des Amnions Theile abgetrennt und am Amnion haftend auf der Körper Oberfläche transplantirt werden." The author just quoted is, I am convinced, mistaken in pushing his views as to the malformation-producing capacities of the amnion so far as this. There can be little doubt that the malformation is atavistic. The English edition of Wiedersheim's 'Comparative Anatomy' * contains the following statement on the subject:—"The occasional existence in men of supernumerary teats and in women of supernumerary teats and mammæ (polythelism and polymastism) is very remarkable. They are usually situated in the thoracic region, and must be considered as an atavism to a characteristic primitive form which possessed numerous teats, and which produced a number of young at a birth. Such a transition from polymastism to bimastism may be seen plainly at the present day in the Lemurs; in them the inguinal and abdominal teats are undergoing a retrogressive metamorphosis, while a single pair of thoracic teats remain well developed. [In *Hapalemur griseus* the single pair of teats is situated on the arm (*Beddard*).] This accords with the fact that most Lemurs bear only a pair of young ones at a time, which they carry with them at the breast." In this connection it is interesting to note that, as Dr. Champneys has shown †, the skin of pregnant women may take on mammary functions in isolated spots, lumps forming in various places, notably in the axilla, from which come (*a*) granular débris like the secretion of sebaceous follicles; (*b*) colustrum; (*c*) milk, expressed from the situation of the sebaceous follicles, as marked by the position of the hairs. These cases show, he says, "that in lying-in women the sebaceous follicles of the skin are capable of producing true mammary secretion. The transition from granular material, through colustrum, to true milk is unmistakable." The observations confirm the opinion that the breast is a highly specialized aggregation of highly specialized sebaceous follicles.

The facts which have just been reviewed leave little room for doubt that accessory mammæ are atavistic and blastogenic.

Hypertrichosis.—Excessive hairiness, which appears to be usually associated with deficient dentition, is an occasionally

* P. 28. † Roy. Med. & Chir. Soc., April 27, 1886.

hereditary condition. Cases of this nature have been recorded by Darwin * and Michelson †. That of Andrian Jeftichew and his son Fedor (*æt.* 3 at the age of description), both of whom had their faces covered with hair "like Skye-terriers," and were nearly edentulous, is a good example ‡. The possibility of this condition having a nervous origin will be discussed in a subsequent section. It is probably atavistic and blastogenic.

Gluteal Protuberances.—The excessive development of the gluteal region amongst the Hottentot women and of their nymphæ may here be mentioned as examples of malformations which have risen to the rank of racial characteristics. They are, apparently, blastogenic in their nature.

In concluding the portion of this paper relating to malformations by excess, the speculation may be permitted as to whether there may not be many hidden abnormalities which are highly hereditary yet which escape notice. Take, for example, the instance of a centrale carpi. One would expect that this abnormality would be hereditary, and possibly it is so; but we have no data to go upon, and in this instance it is not probable that we ever shall have. In other cases, however, a little trouble on the part of the observer might enable many gaps to be filled up. As an example of what I am alluding to, I may cite a curious case, the only one known to me of a described hereditary abnormality in the arterial system, though it is probably by no means an isolated case, were the question to be carefully investigated §. The radial artery in a male, on both sides passed over the supinator longus at from 3·0 cm. to 4·0 cm. above the wrist, and ran over the radial extensors above the styloid process to its normal distribution. All this man's children possessed the same abnormality on the left side, the daughters transmitted it better than the sons, and amongst the grandchildren it was met with on both sides on four, on one side only in four, and was absent in seven.

Part 2.—*Malformations by Defect.*

In this section the malformations by defect will be discussed, and at the outset it may be said that their etiologies in many

* 'Animals and Plants under Domestication,' vol. ii. p. 320.

† "Zum Capitel der Hypertrichosis." Virchow's Arch. Bd. 100, S. 66.

‡ Lancet, 1873, ii. p. 613; and Virchow, Berl. Klin. Woch. 1873, No. 29.

§ Chicago Med. Journ. & Exam. 1879, p. 475.

cases offer much greater difficulties than those of the group just concluded.

Dwarfs.—The arguments which were used to show that giantism was a blastogenic condition due to excess of formative material, might, in converse, be employed to prove that dwarfishness is blastogenic and due to deficiency of material. As with giants, so with dwarfs, sterility is the rule. In their case, we have on this point even better evidence than in that of giants, since dwarfs have been made the subject of direct experiment. "Cathérine de Médecis s'amusa, dit on, à rassembler des nains des deux sexes et à les marier. L'électeur de Brandenbourg répéta la même expérience; ils n'eurent pas d'enfants. Geoffroy St.-Hilaire ne découvre qu'une seule exception, celle du nain Borivilaski; mais sa femme était de taille ordinaire et fut suspectée" *. Though sterility is the rule, yet there are indications of hereditary influence in this group also.

Thus, as St.-Hilaire points out †, in the greater number of cases the same mother has given birth to two or more dwarfs, amongst the cases he cites being that of the dwarf brother and sister described by Aldrovandus ‡. I have also met with an account of one case where dwarfishness was distinctly hereditary §. Francesco Leporatá, born of full-sized parents, was at the age of 83, 1·130 mtr. He had married a full-sized woman and had issue at the time of enquiry:—(1) *Doralice* (F.), æt. 50, height 1·130 mtr.; (2) *Anna*, æt. 41, height 0·980 mtr.; (3) *Maria*, æt. 31, height 1·155 mtr.; (4) *Maddalena*, æt. 34, of normal height; (5) *Antonio*, æt. 44, height 1·340 mtr.; (6) *Pietro*, æt. 42, height 1·300 mtr. Antonio (5) married twice. His first wife, who was of full size, bore him a normal-sized daughter; his second, also a full-sized woman, bore him three sons:—(*a*) æt. 14, height 0·945 mtr. (normal for age, 1·49 mtr.); (*b*) æt. 9, height 0·970 mtr. (normal, 1·219 mtr.); (*c*) æt. 7, height 0·910 mtr. (normal 1·106 mtr.). Pietro (6) married a full-sized female, and had issue—(*a*) M. æt. 6, height 0·825 mtr. (normal 1·046 mtr.); (*b*) F. æt. 3, height 0·650 mtr. (normal 0·864 mtr.); (*c*) M. æt. 1½, height 0·616 mtr. (normal 0·744 mtr.).

* Topinard, *op. cit.* p. 436.

† *Op. cit.* p. 159.

‡ 'Monstrorum Historia,' pp. 603 & 604.

§ Quoted from Ludwig Frank, Mem. della R. Accad. di Torino, t. 25. p. 96 (Torino, 1820), by Taruffi, Riv. Clin. 2 ser. viii. p. 33; Abstr. Schmidt's Jahrb. t. 198. S. 8.

Absence of Bones or Parts of Bones.—Malformations of this kind are not very uncommon. In the following, amongst other cases, the condition has been hereditary:—(1) Prof. Sir Wm. Turner has described * a case of shortening of the ring-finger, which was 1·2 in. shorter than the middle, and only 0·5 in. longer than the little finger. This condition was not due to absence of any of the bones, but to imperfect growth of the metacarpal bone. In the family was also present widening of the great toe and thumb—sometimes with short ring-finger, sometimes without. The malformation ran through six, or possibly seven, generations. (2) In another family † shortening, due to absence of phalanges, in a variable number of digits ran through three, or possibly four, generations. (3) Absence of patellæ through two ‡ and three § generations has been recorded. (4) Imperfection of one or other end of one or both clavicles has existed through three generations in a family ‖. There can be little doubt that these malformations are blastogenic, and due to deficiency of formative material.

Peromelia and Perodactyly.—Absence of parts or of an entire limb or digits. This group opens up the whole question of intra-uterine amputations, which requires some little consideration. First described by Montgomery ¶, it has been discussed by a number of later writers, some of whose opinions must here be dealt with. In Montgomery's first case the foot which had been amputated was found in the membranes, and was apparently two months younger than the child to whom it belonged. In another case the constriction had severed everything but skin and bone. According to his view inflammatory lymph was thrown out, which constricted the growing part and effected the amputation. Dr. Macan ** has described a case of amputation of the arm below the insertion of the biceps, in which the cicatrix was quite healed. The amputated part was not found, though searched for. In 30 years only one case of intra-uterine amputation had been recorded in the practice of the Rotunda Hospital, Dublin. In the discussion which followed upon the paper in which this case was described, Dr. Kidd said that in something less than 30 years he had seen

* Journ. Anat. & Phys. vol. xviii. p. 463. † Tilley.

‡ Med. Notes and Reflections, p. 33.

§ Med. Gaz. 1833, p. 519. ‖ Warren Museum Catalogue, no. 217.

¶ In an Essay "On the Spontaneous Amputation of the Fœtal Limbs in Utero," at the end of his "Signs and Symptoms of Pregnancy."

** Dubl. Journ. Med. Sci. vol. lix. p. 55.

four cases. In one of these, the leg was amputated between the knee and the ankle, the severed portion being found in the membranes. Other bands had nearly amputated some of the fingers. According to Hennig *, the substance which forms the bands may be formed sometimes from the skin of the embryo, sometimes from the membranes, and sometimes from both. The skin alone was, in his opinion, the cause in most of the recorded examples of spontaneous amputation. Before considering what proportion of the cases of defective limbs and digits may be due to this cause, it will be necessary to consider two special classes of cases. (1) Cases of perodactyly with absence of a forearm-bone and carpal bone. I have described † an instance where radius, scaphoid, trapezium, and thumb were all absent on both sides. It is obvious that cases such as this could not be produced by amniotic causes, but are due to absence of formative material and blastogenic in their nature. (2) There is a curious group of cases where on the end of the stump are to be found fingers generally in an imperfect state of development. Or there may be a want of the intermediate parts in the extremities so that the hand is attached immediately to the shoulder and the foot to the hip, as in the remarkable case of Marco Catonze, figured by Vrolik ‡ and Förster §. Simpson ||, in 1841, noticed what he called a tendency to rudimentary reproduction of the amputated members on the face of the stump. This he compared with the cases of reproduction of limbs in lower animals. Sturge ¶ describes an interesting case of a man aged 22, in whom the radius and ulna on the left side ended in a conical stump 3 in. below the elbow-joint. On the flexor aspect of the stump, and situated transversely across it, were five little projections, the one nearest to the radial side being the largest and the remainder gradually decreasing to that on the ulnar border; the largest and the adjoining one had well-marked nails. The author's remarks upon the subject will be more appositely quoted somewhat later. The point is a very interesting one and by no means clear, but it may be doubted whether there is really any truth in the restoration theory, and whether the facts are not much better explained by some such hypothesis as that offered

* "Ueb. d. Nebenbänder u. Schafhautstränge in der Eihohle d. Mensch.," Virchow's Arch. Bd. xix. S. 200.

† Anat. Anzeiger, Jahrg. 3. S. 63.

‡ Art. "Teratology," Todd's Cycl. of Anat. & Phys. figs. 624, 625.

§ *Op. cit.* Taf. xi. figs. 6 & 7.

|| Selected Obstetric Works, p. 129.

¶ Trans. Path. Soc. vol. xxxi. p. 208.

by the last-named writer, when he says "they [*i. e.* the imperfect digits] represent the amount of vitality left in the embryonic cells from which the extremities of the limb should have developed." We explain the presence of extremities on sacral tumours representing an imperfect parasitic twin, such as those of Anna Maria Przesomyl and the other given by Braune *, by supposing that the cells forming that portion of the limb have come to development, whilst others have failed to do so; and there seems no reason why a similar explanation should not be given in the cases under consideration. Passing from these special groups, it may next be mentioned that Erlich † has grouped congenital defects of extremities into the following divisions:—(1) deficiency of formative material (Anlagekeim); (2) deficiency of division or segmentation (Gliederung) where a ray (Tibia, Radius, with tarsals or carpals, &c.) is absent; (3) deficiency of growth from injury to cartilage or bone, Fœtal Rachitis; (4) spontaneous amputation, or strangulation.

We have now to consider the bearing of the facts just detailed upon the section of the subject now under discussion. First of all we may dismiss, I think, the subject of peromelia, since I am not aware that it has ever been described as hereditary in man. St.-Hilaire ‡ has given an account of one case in which it was hereditary in dogs, and I know of no other of any kind. Perodactyly is, however, not uncommonly hereditary. In a case given by Fotherby §, the hands and feet were both affected, only great and little toes being present in the latter; the malformation ran through five generations. Other cases have been given by Holmgren‖, five generations; Krabbe¶, three; and Lucas, four**.

In considering the nature of this defect it should be borne in mind that there are several preliminary conditions, so to speak, leading up to it and often merging into or blending with it, none of which could be caused by amniotic agencies. Thus Annandale gives the following degrees of union of digits, a condition

* 'Die Doppelbild. u. angeb. Geschw. d. Kreuzbeingegend,' Leipzig, 1862, Taf. iii. figs. 1, 2, 3, & 7.

† "Untersuch. üb. d. Cong.-Def. u. Hemmungsbild d. Extrem.," Virch. Arch. Bd. 100. S. 107.

‡ *Op. cit.* vol. ii. p. 570.

§ Brit. Med. Journ. May 22, 1886.

‖ Upsala läkarefören. förhandl. xvii. 7 og 8, S. 513; Abstr Schmidt's Jahrb. Bd. 196. S. 121.

¶ Nord. Med. Ark. xii. 20. S. 1; Abstr. Schmidt's Jahrb. Bd. 196. S. 121.

** Traité de l'Hérédité Naturelle (Paris, 1847), p. 198.

recognized as leading up to perodactyly *:—"Two or more digits may be united—(1) by loose folds of skin only (the true webbed condition)†; (2) by a more intimate connection of the skin and deeper soft tissues; (3) by the union or fusion of the bones as well as the soft textures. Besides these forms of union, the digits of one hand or foot, or of both hands and feet, may be all massed together into one lump, so that it may be almost impossible to distinguish the individual fingers or toes." This gradation of defects, the minor ones being of a kind not assignable to amniotic agencies, is the first point in favour of the blastogenic nature of the defect. A second is the nature of the defect itself. Whoever will take the trouble to examine the figures in Förster (Taf. xii. figs. 1–21) will not have much difficulty, I think, in deciding that they are highly unlikely to have been due to amniotic agencies. Many of the absent digits are central, yet these are surely more likely to escape entanglement in loops of fibrin than the marginal, which so frequently remain as the sole representatives. I may here quote *in extenso* Sturge's remarks to which I have before alluded. Speaking of his case, which he looks upon as one of intra-uterine amputation, he says:—"The congenital deficiency may also be due to (1) a primary inherent abnormal condition of the ovum whereby its healthy development is interfered with in one or more directions; or (2) a localized morbid condition of one or more parts of the embryo at some time after conception. The former of the two conditions must be invoked in explanation of many cases of monstrosity, in cases where there is congenital hypertrophy of parts, and it is the most probable explanation of supernumerary parts. There can, therefore, be no reason why it should not produce the opposite condition, viz. congenital atrophy of limbs and congenital absence of portions of limbs. On the other hand, an inflammatory condition capable of throwing out organized lymph in sufficient quantity to produce intra-uterine amputation is quite as likely, if it attacks that portion of the blastoderm from which a limb is developed, to kill the delicate embryonic cells, or to modify their nutrition so profoundly that their subsequent development will be gravely compromised. I think it is worthy of inquiry whether rudimentary fingers have been present in any of those cases where the amputated limb has been

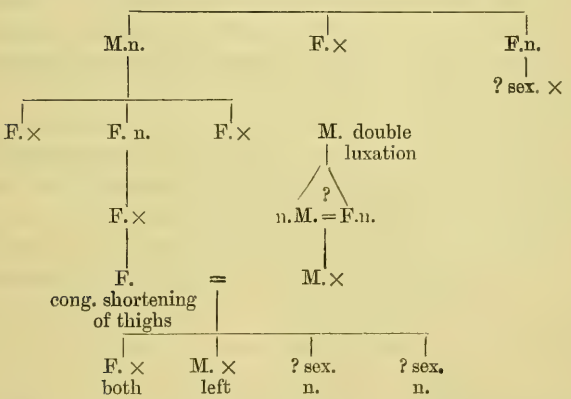
* *Op. cit.* p. 46.

† This may be hereditary. It has been described as passing through four generations. Edinb. Med. Journ. 1858–9, p. 501.

found. It is very easy to account for them on the hypothesis of mal-development, for in that case they represent the amount of vitality left in the embryonic cells from which the limb should have developed. On this hypothesis, we should expect to find, as in fact have been found, many degrees of development ranging from minute nodules, representing fingers at one end of the scale, up to extremities of limbs which differ but little from the hand at the other end."

To conclude this group, without excluding the possibility of cases of perodactyly, originally caused by amniotic bands being hereditary, since there are no data for such a denial, it appears to me, for the reasons given above as well as from those which were brought forward when dealing with the subject of polydactyly and amniotic bands, that on the whole it is far more probable that perodactyly is nearly always due to defect of material, and is therefore blastogenic.

Congenital Luxation of the Femur.—This is a condition which requires some consideration, since it is certainly at times hereditary and since its origin has been very diversely explained by different writers. I append the following hereditary case, doubly remarkable since present on both sides of the house *:—



Professor Bennett, in an address on this subject †, enumerates the following opinions which have been expressed as to its cause. Dupuytren considered it to be due to an original fault of the germ, which, as Bennett says, is "sufficiently vague." Stromeyer attributed it to a disproportion between the head of

* Quoted by Dupuytren from Massiat, Med. Gaz. 1833, ii. p. 570.

† Dubl. Med. Journ. lxxix. p. 11.

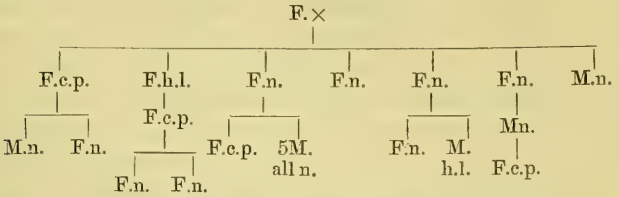
the femur and the acetabulum, Cruveilhier and Chelius to the position of the fœtus *in utero* or to violence in delivery, Ammon to arrest of development, Guerin to irregular nervous action, and South to an abnormal method of delivery. Recent writers, he proceeds, have assumed that the last-mentioned cause is always the correct one. This, however, is not the case; the specimen which he himself dissected was perfectly normally delivered by a head presentation. Other observations point to the fact that one of the causes, if not the chief cause, is a failure of development of the acetabulum due to a lack of material. Shaw, describing a living specimen, states that on digital examination, "a projecting irregularly shaped surface, supposed to be the imperfectly-developed acetabulum, was felt" *. Shepherd † has described a case which he had the opportunity of dissecting. The female, aged about 50, had the right leg smaller and shorter than the left. The wing of the right ilium was thinner, more upright, more curled inwards, and smaller. The acetabulum was a mere triangular depression in the bone with its apex directed upwards and to the right and its base corresponding to the cotyloid notch. The edges of this triangular depression were smooth and curled inwards and but slightly covered with fibro-cartilage. Its measurements were 2 in. long, $\frac{3}{4}$ in. broad, $\frac{1}{2}$ in. deep. Grawitz ‡, in a paper on the subject, refers the malformation to a failure of development of the Y-shaped cartilage. In twelve cases which he examined, there was no trace of any inflammation of the joint as the cause. Whilst believing that a defect of development is the chief cause, the possibility of irregular or excessive nervous action being also a factor must not be overlooked, since the improper action of the muscles arising from it may induce the displacement of the head of the femur from the imperfectly developed acetabular cavity. The defect will, if due to defective development, be probably blastogenic in its nature. There is, however, just the possibility that mal-nutrition of the fœtus *in utero* may be the cause. This is a large subject, which will receive the attention it deserves in a separate and subsequent section.

* Trans. Path. Soc. xvii. p. 206. The author remarks that it has never been explained why the malformation should so often affect both acetabula and occur more frequently in females.

† Journ. of Anat. & Phys. vol. xiv. p. 368.

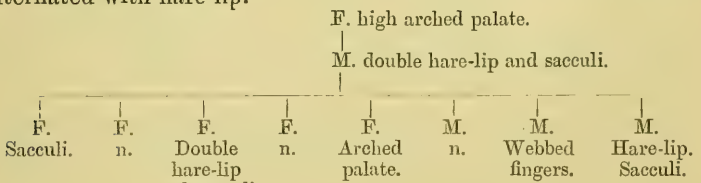
‡ "Ueber die Ursachen der angebornen Hüftgelenkverrenkungen," Virch. Arch. Bd. 74. S. 1 (1878).

Cleft-Palate and Hare-Lip.—These malformations are undoubtedly hereditary. Fritzsche found the factor of heredity to exist in five out of fifty-two cases coming under notice in the Zurich Klinik in ten years*. Mason† has recorded others, and Biondi‡ gives the following table from Passavant§:—



(h.l.=hare lip; c.p.=cleft palate.)

I have mentioned these numerous cases since Förster and others have inclined to the opinion that cleft-palate is not hereditary. Before leaving the question of heredity in connection with these defects, it should be mentioned that the parents, if not affected with the lesion itself, may present minor malformations tending in that direction. Thus Mason (p. 18) says:—"Sir William Ferguson used to look for, and generally find, a partial defect in the upper lip and jaw of one or both of the parents, and since he directed my attention to this point, I have observed it in many instances." Again, Knaggs‖, in a report of 660 midwifery cases conducted in Australia, describes one of exaggerated hare-lip and cleft-palate, in which several individuals on the mother's side were similarly affected. Moreover, "the mother possessed a very high-arched palate, so much so as to create the impression that she had narrowly escaped the deformity itself." Finally, Murray¶ has placed on record a case in which other malformations of the same region co-existed with or alternated with hare-lip.



* 'Missbild. d. Gesichts.' (Zürich, 1878), p. 6.

† 'On Hare-Lip and Cleft-Palate' (Lond. 1877), pp. 21 & 64.

‡ "Lippenspalte," Virch. Arch. Bd. 91. S. 173. § Arch. f. Heilk. 1862, p. 305.

‖ Dubl. Med. Journ. lxxi. p. 431. ¶ Med.-Chir. Rev. vol. xxvi. p. 502

When we come to inquire into the causation of these defects, we find various theories given to account for them. Virchow *, writing of branchial and other clefts, says that without particularizing whether traumatic, thermic, or other causes act, the main fact is that the defects arise from an irritative process. Some may call this inflammatory; in any case it is not passive but active. Some forms of palatine and facial clefts have, he thinks, a similar origin in an early inflammation. It should be mentioned that this was written as long ago as 1855, and that the theory which accounts for many defects by fœtal inflammation has lost much ground since then. The defect may be due to deficiency of material, and this again, it seems possible, may be due to mal-nutrition by the mother. I quote the following passage from Oakley Coles's interesting chapter on the etiology of cleft-palate, to which I shall have to recur in a later section †:—

"Dr. Ogle has called attention to the fact that 99 per cent. of the lion-cubs born in the London Zoological Gardens have cleft-palates, and he has referred this curious phenomenon to the artificial diet necessitated by the enforced captivity. It has, indeed, been contended, in reply to this theory, that the experience of the London Zoological Society is exceptional, differing from that of other menageries, and Mr. Pollock ‡ has suggested that we must seek for the cause of the phenomenon amongst other conditions than the food-supply. It is true that among the lion-cubs born in the Dublin Gardens, cleft-palate is seldom noticed; but it is stated that it used to occur quite as frequently as in London, when the feeding was conducted in a similar way, viz. by supplying only the meat of large animals. Now, however, that the lions are given goat twice a week, which they can eat bones and all, the proportion of cleft-palate has become quite insignificant. These observations seem to point to the possibility of cleft-palate in the human subject being due to an analogous departure from a natural diet amongst civilized nations, but it is at all times perilous to argue from the lower animals to man. At any rate the evidence at present before us does not admit of anything more than conjecture." If the in-

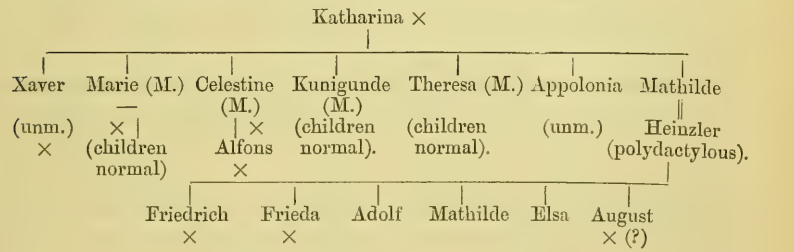
* "Ueb. Missbild am Ohr u. im Bereiche des erstens Kiemenbogens," Virch. Arch. Bd. 30. p. 221.

† 'Deformities of the Mouth' (Lond. 1887), p. 37.

‡ Holmes's System of Surgery, vol. iv. p. 420.

flammatory theory or that of mal-nutrition be true, the condition would be somatogenic in its nature. Those cases where the cleft is due to the presence of a tumour are undoubtedly so, but they are, I believe, never hereditary. On the other hand, cases due to an original lack of formative material would be somatogenic. It is possible that these and some other defects may be due to a hitherto little recognized cause, that is a failure of nerve-action during intra-uterine life. I purpose devoting a section at a later part of this paper to the consideration of this hypothesis. For the present, however, the subject of cleft-palate must be left in a highly undecided state.

Edentulism.—Partial absence of teeth, accompanied or unaccompanied by peculiarities of the hair, is an hereditary condition at times. Cases have been given by Darwin and others, and my friend Mr. Humphreys was good enough to supply me with notes of some observed by himself, which I published in my paper on "Congenital Malformations and Heredity." One very curious case communicated to me by Dr. Fackenheim, of Eisenach, and since published in his paper already quoted, I shall give at length, because of its bearing upon the question of mal-nutrition as a cause of defects, which has yet to be dealt with. In the case I allude to there is on the father's side polydactylism both of the hands and feet, on the mother's anomalous dentition. The mother's genealogy, and the children born of her union with the polydactylous Heinzler, stands as follows:—



Katharina, the grandmother, had only two upper pointed teeth in place of the incisors, and two molars. Her children were situated, as regards their teeth, as follows:—Xaver, unmarried, is somewhat similar to his mother, so is Marie, who has normal children. Celestine is similarly affected, and so is her son Alfons, who possesses only four teeth including one first

molar. Kunigunde and Theresa, as also their children, have the normal dentition; so also has Appolonia, who is unmarried. Mathilde has six exceedingly large and pointed teeth like canines, with wide gaps between, which represent the incisors, the remainder of her dentition being normal. Mathilde married Heinzler, in whose family polydactylism is hereditary. Of their children, Friedrich (æt. 11) has the most singular dentition of his generation. The lower incisors are in his case completely absent; in place of the upper incisors are two conical teeth. The remaining teeth are normally formed. The alveolar border of the inferior maxilla is thin and defective in the situation of the defective teeth. This is less marked in the superior maxilla. There is no trace of a frænum linguæ, but otherwise the mouth is quite normally formed. It should be noted that these teeth belong to the milk-dentition, no permanent teeth having appeared. The defect is already commencing to exercise a marked effect upon the contour of the face, and undoubtedly influences the speech. The sister Frieda (æt. 14) is not quite so deficient as her brother, but the separate teeth are not so well formed as his. Like the brother, she still possesses her milk-dentition. In the upper jaw, in place of the incisors, are two pointed or crescentic teeth with their apices towards the middle line. In the lower jaw the incisors are replaced by three irregularly conical teeth, the central one being somewhat behind the other two. The remaining teeth are fairly normal, but have considerable gaps between them. There is a very small frænum linguæ, but no other oral abnormality. In both children the teeth made their appearance at the proper time. Both of these children inherit also the polydactylism of the father. They are thoroughly healthy, have never suffered from any severe illness, and have no abnormal condition of the hair. The alveolar processes of the youngest child, August, are thin, from which the mother, reasoning from what she saw in the other children, confidently asserts that he also will exhibit similar abnormalities. Dr. Fackenheim finally points out that the rudimentary condition of the alveolar processes in these children renders it impossible that the missing teeth are retained in the jaw. No one, so far as I am aware, has ever advanced the theory that defect in the number of teeth in a parent, due to the labours of the dentist, can be transmitted to the children. The fact that this experi-

ment, tried upon so large a scale, returns a negative reply is indeed to my mind one of the best arguments against the transmissibility of mutilations. There can be little doubt that the recorded cases of congenital and hereditary edentulism (congenital in the sense of the impotentiality being so) are blastogenic in their nature.

Microphthalmus.—This is the first of a group of malformations relating to the eye. In dealing with them, I have to express my obligations to my friend Mr. Priestley Smith, whose extensive knowledge of ophthalmological subjects has supplied my deficiencies in that direction. Microphthalmus is an hereditary disease—Sedgwick * narrating a case where it was hereditary on the maternal side and deaf-mutism on the paternal, both defects co-existing in some of the unfortunate descendants. It is extremely interesting in this connection to see how opinion as to the cause of microphthalmus has changed of late. It was thought by some authorities, for example Deutschmann †, that a fœtal inflammation was the cause. Were this true the condition would be somatogenic. Quite recently, however, Hess ‡, after a very careful examination of six microphthalmic eyes, has concluded:—(1) that there was no sign of past or present inflammation to be discovered; (2) that a union existed between the vitreous and the outer tunics of the secondary optic vesicle effected by means of a tissue nourished by the hyaline artery or a representative of that vessel. He goes on to state that he cannot regard this as being in any way an inflammatory product, but considers it to be the result of an atypical embryonic development of the intruded mesoblastic layer which goes to form the vitreous. He refers to other published cases which resembled his in important respects, and to which he is inclined to attribute a similar causation. The connective-tissue band formed in these microphthalmic eyes may possibly be related to the funiculus scleræ, described by Hannover and shown by Rotholz to be the permanent representative of a structure existing in fœtal life. Should these observations be correct, as seems highly probable, the malformation is a blastogenic one, though it is perhaps somewhat doubtful as to which class it should be referred to.

* Med.-Chir. Rev. vol. xxviii. p. 205.

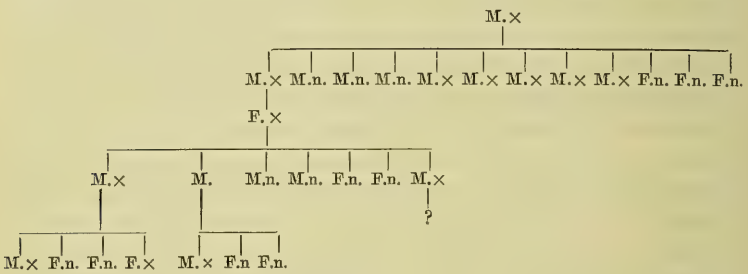
† Klin. Monatsbl. f. Augenheilk., March 1881.

‡ v. Graefe's Arch. f. Ophthalm. xxiv. 3.

Absence of Iris.—Page * has recorded a case where this condition, combined with microphthalmus and nystagmus, existed in a mother, two daughters, and a grand-daughter, two sons and a daughter having escaped. Sedgwick † quotes another where absence of iris ran through three generations. There can be no doubt that this is a blastogenic condition.

Coloboma.—A failure to close completely on the part of the choroidal fissure, doubtless blastogenic in its nature, though whether due to defect of material or to a nervous cause is not certain. It is hereditary, and Darwin ‡ has given a case where it passed through four generations.

Congenital Dislocation of the Lens.—I give here a table, drawn up by Stanford Morton §, where this curious condition was hereditary.



This defect "is sometimes associated with coloboma of the choroid or optic disc, sometimes with persistent hyaloid artery; more frequently it is met with in eyes which appear otherwise healthy. A partial lateral displacement indicates a faulty development of the suspensory ligament, by reason of which the traction upon the lens is unequal at different parts of its circumference. The displacement is usually upwards or upwards and outwards, indicating a defect in the lower part of the ligament, analogous to an imperfect closure of the fœtal slit. It is usually present in both eyes, and is symmetrical or nearly so in the two. It has frequently been met with in several members of one family and in successive generations" ‖. The defect from this appears to be one of deficient development, blastogenic in its nature.

* Lancet, Aug. 8, 1874.
† From Ammon's Zeitschr., vol. i. no. 4.
‡ *Op. cit.* vol. i. p. 454.
§ Oph. Hosp. Reps. ix. 435.
‖ P. Smith, in Heath's Dict. of Pract. Surg., Art. "Crystalline Lens."

Strabismus.—Squint is sometimes hereditary, though Mr. Priestley Smith tells me very rarely. Portal *, in his "Considerations sur les Maladies de Famille," describes an imperfect form, called the Montmorency sight, with which nearly all the members of that family were affected. In speaking of squint, it must be very carefully borne in mind that the only true congenital cases are those which are noticed instantly after birth. This sounds like a truism, but it is a very necessary caution, since there are many cases noticed, not at birth, but within the first few weeks of infancy, which are called, incorrectly, congenital. These last-mentioned cases are secondary in their nature and follow upon hypermetropia, which is very commonly hereditary. As regards the true hereditary cases, Welcker and Landolt † remark that they probably result always from some lesion of the nerve-centres or of the motor-oculi nerve in intra-uterine life. The muscles corresponding are then rudimentary or present abnormal insertions, as in a case recorded by M. Henck, who had the opportunity of making an autopsy on a child thus affected ‡. I am informed, I should say, that the question as to whether the muscles are really shortened in this affection, is one which is much disputed by ophthalmologists. As regards the primary hereditary hypermetropia, which is often the cause of the secondary strabismus, since the hypermetropic eye is smaller than normal, a deficiency of material may be the factor which produces it, or, and I think more probably, the defect may have a nervous origin. As regards the opposite condition of hereditary myopia, Mr. P. Smith writes § :—"Firstly, there is the hereditary predisposition. Different observers estimate the importance of this factor very differently, but hardly anyone will deny that under similar circumstances, the children of myopic parents are more liable than others to acquire myopia, and that this fact is a weighty one in relation to the general progress of the disorder through successive generations. Whether the transmitted tendency depends chiefly on peculiarities in the tissues of the eyes themselves, or on the mechanical relations subsisting between the eyes on the one hand, and

* *Cf.* Ribot, "Heredity," Engl. transl. p. 39.

† Traité compl. d'Ophthalm. vol. iii. p. 867.

‡ "Ueber angeb. vererb. Beweglichkeits-Defect der Augen," Klin. Monats. i. 1079.

§ Ophthal. Rev., June 1886.

the muscles, the optic nerves, and the orbits on the other, is not yet positively known. Secondly, there is the supposed correlation between the growth of the brain and the growth of the eye, by reason of which a high degree of cerebral development is apt to be associated with an overdevelopment of the eye." (The remaining two causes given in the paper quoted from are not germane to the subject of hereditary defects.) The consideration of the defects dealt with in this last section will be better deferred to the section in which the influence of nerve-action is discussed.

Absence of the External Ear.—Sedgwick * gives a case of absence of the left external ear—a father not himself presenting the defect, had a son who did; the father's cousin, a male, was affected as were two of his male children, a daughter escaping. This case, as he remarks, seems to point to the existence of the defect in some earlier ancestor. The same writer quotes a case recorded by Anderson Smith † in which a woman, two of her daughters, and two grand-daughters, had rudimentary lobules to their ears, the male children and grand-children being normal.

Cleft Lobule of the Ear.—This condition is of particular interest, since it was first brought into prominence by being advanced as an example of the heredity of a mutilation, in opposition to Weismann's views. So far from this being the case, the defect, now that attention has been directed to it, seems rather to support his contention. It is to be hoped that the stimulus to observation given by the publication of Weismann's Essays may lead to the clearing up of more of the many vexed questions in the field of Teratology. The history of the controversy on this condition is as follows. Dr. Emil Schmidt ‡ described a case in which the mother had acquired a cleft of the lobule of the left ear by the tearing through it of an earring whilst at play, at the age of 8 years. Of her eight children, the second, a boy, presented a cleft of the lobule of his left ear, which was regarded by Schmidt as an inheritance of the mother's mutilation. However, His § and Weismann ‖ have both pointed out that the cleft in the son's ear is quite different from that of the mother, and occupies a different position. Finally,

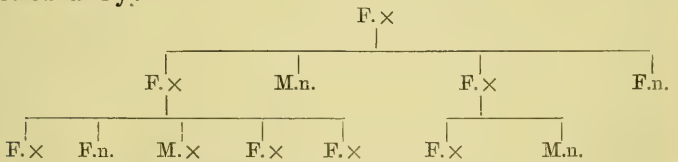
* Med.-Chir. Rev. vol. xxviii. p. 206. † *Ib.* vol. xxxi. p. 457.

‡ "Ueb. Vererbung individ. erworb. Eigensch.," Corresp.-Bl. d. deutschen Ges. f. Anthrop., Nov. 1888. § *Ib.* March 1889.

‖ 'Ueb. d. Hypothese einer Vererb. v. Verletzungen,' Jena, 1889.

Dr. Oscar Israel has published a paper * on the condition, in which he describes two similar cases in which there is no mutilation in the parents. Moveover, he points out that the cleft always has its site in the position of a fissure of normal occurrence in the development of the ear, which fissure has been called by His "Sulcus intertragicus." The malformation, therefore, falls into line with the other congenital clefts as an arrest of development.

Branchial Fistula.—A failure in the closing of the branchial clefts producing the condition known as branchial fistula is at times hereditary. In fact the influence of this factor appears to be considerable, since Heusinger † was able to trace its action in seventeen out of forty-six individuals. The following table given by Ahlfeld from Ascherson ‡ gives a good example of an affected family:—



The condition seems to be undoubtedly blastogenic, though whether due to lack of material or to nervous causes is not clear.

Hypospadias.—This malformation is sometimes hereditary. Lucas § gives a case in which grandfather and father both were affected; whilst the son had the same defect combined with atresia ani and rectum opening into the urethra. In another case, alluded to by both Darwin and Lucas, and originally described by Meckel ||, a female in whose family hypospadias was present, gave birth to two sons, both affected with the same deformity.

This exhausts the list, so far as I am aware, of hereditary malformations of the second sub-division. The last division, that of abnormalities not due to faults of formative material, which must next be dealt with, will not require any very lengthy consideration.

* "Angeb. Spalten d. Ohrläppchens," Virch. Arch. Bd. 119. S. 241. [Since this paper was written, several communications have appeared on the subject, the titles of which are here given for the sake of completeness:—Ornstein, Arch. f. Anthrop. t. xviii. f. 4; v. Swiecieki and His, Arch. f. Anat. u. Entw., Jahrg. 1890, Hft. 5 & 6; cf. also Laloy, "Malform. Héréd. du Pavillon de l'Oreille," L'Anthropologie, i. p. 5.—*Note*, Dec. 30, 1890.]

† Virch. Arch. Bd. 29. S. 358.

‡ De fistulis colli congenitis, Diss. pro venia legendi (Berlin, 1832).

§ (From Dr. Ritter von Rottembourg,) vol. i. p. 324.

‖ Hdb. d. path. Anat. vol. i. p. 20.

Part 3.—*Malformations possibly Mechanical.*

It will be most convenient to take this group under the headings indicated in the first section.

Clefts due to Tumours.—Fœtuses born with such deformities are, I believe, almost always still-born. In any case I have never met with an account of any case where such a condition was hereditary.

Clefts due to Fluids of an Inflammatory Nature.—As examples of this may be mentioned such defects as abdominal fissure, sufferers from which are always still-born; anencephaly again, a condition in which life is impossible for more than a few hours at the most; with, possibly, spina bifida. Opinions are, however, divided on this question. "Which is the primary defect," says Mr. Treves *, "arrest of development in the bones, or dropsy of the membranes? Does the deficiency in the bony canal encourage a protrusion of the membranes? Or has the protrusion prevented the proper formation of the osseous canal?" To this question, he says, no satisfactory answer has yet been given.

Defects due to Formation of Inflammatory Fibrous Tissue.—That part of this section which relates to deficiencies of extremities has already been sufficiently dealt with in the paragraphs on Peromelia and Perodactyly, since most of what was there said in connection with the influence of amniotic bands in the production of defects might also be applied to the present question. As far as atresia ani is concerned, if any cases are attributable to the cause under consideration, that defect is of no importance so far as this inquiry is concerned, since there is no evidence, so far as I am aware, that it is ever hereditary.

Defects due to Amniotic Pressure.—These defects are of a totally different nature to those previously discussed in connection with the amnion. The defects of this class are due to the compressing action of an amnion free from obvious inflammation or other disease. This pressure may follow, it appears, from (1) a deficiency of liquor amnii, whereby the fœtus is brought too much under the influence of the amnion and possibly also of the uterine walls; (2) a want of sufficient growth on the part of the amnion itself, whereby the growing embryo is compressed; (3) possibly, the abnormal size of the

* Internat. Encycl. of Surgery, vol. iv. p. 891.

child itself. It is, however, unlikely that this factor, save perhaps in very rare cases, can produce any lasting effects. Every obstetrician has seen large children with their feet "tucked," to use the common phrase, but which have suffered no permanent injury. The malformation, however, which does require some consideration here is that of Talipes, which is at times hereditary, and which is thought by some to be caused by amniotic pressure. I shall, I think, give the best idea of opinion on this subject by some extracts from a very careful monograph on the question by Parker and Shattock *. With regard to nerve causes which have been considered by some to be the most probable solution, they say:—"By those who advocate this theory it is argued, that because talipes ensues after recognized nerve-lesions, a nerve-lesion must therefore be the cause of those forms of talipes which are congenital, since the two deformities are so outwardly alike. This argument, they think, is strengthened by the fact that talipes is often associated with malformations of the nerve-centres (such as anencephalism, spina bifida, &c.). In the first place, the supposed nerve-lesions have never been demonstrated. Apart from the fact that in our own case the nerve-centres and the nerve-trunks were perfectly normal, there are clinical facts which tell against the nerve theory of causation. The most important of these is that talipes is an accidental, and not an essential, sequel of paralysis .... If further evidence in this direction be needed it is to be found in the fact that congenital malformation of the nerve-centres occurs without the association of talipes, as is abundantly shown by the specimens of anencephalus preserved in museums; Mr. Pepper has recorded † a case of so-called complete absence of brain and cord associated with talipes calcaneus of both feet, among other deformities. In this case we should have to assume a negative action of the nerve-centres, a want of nerve-control, perhaps, if we would invoke a nerve-cause at all .... We think it quite possible in a few cases that there may be a nerve-lesion apart from such manifest conditions as spina bifida, &c. For very occasionally at the time of birth (and the same may be found at comparatively early ages in the fœtus) the limb is more or less atrophied, a condition which is especially observable when the

* Trans. Path. Soc. vol. xxxv. p. 423.

† Med. Press and Circ., May 8, 1878.

atrophy is confined to one side. We must, however, confess that we have no direct observations on the nerve-centres in support of these views. They are derived solely from clinical observations of cases, and the results may very well be put down as due to mechanical causes, and belong to the same category of cases as intra-uterine amputation &c." In conclusion, they say:—"We think that of all the explanations of club-foot hitherto offered, a mechanical one is the most reliable and satisfactory for the great majority of cases." Their remarks on heredity in connection with this malformation are also worth quoting. "With regard to heredity, the whole question is so obscure that it is hardly profitable. Although cases of hereditary transmission of the deformity from parents to children do undoubtedly occur, yet in the vast majority of instances no such hereditary influences can be traced, and this is even true in instances where several children in the same family have suffered. The influence of heredity, however, may be invoked with equal force, whatever view of the pathology of the disease be adopted. But at first sight it may seem not a little remarkable, that in some cases the deformity is transmitted along the paternal line; and it may be difficult to harmonise this fact with the influence of environment on which we have insisted. It need only be remarked, however, that the environment of the fœtus depends upon the fœtus itself, not less than upon the mother. For most recent observations show that the liquor amnii may be considered throughout a fœtal and not a maternal product. Excess or deficiency in its amount may, therefore, be the result of a tendency inherited either from the father or the mother." It is evident that no definite opinion can at present be hazarded as to the cause of talipes and, therefore, as to its nature, whether blastogenic or somatogenic.

Deficiencies due to Amniotic Adhesions.—These have already been discussed sufficiently in earlier sections.

Having now passed in review all the congenital malformations with which I am acquainted, it remains for me to consider in the succeeding sections certain general questions which appear to need a separate treatment. These include, *inter alia*, the effects of maternal impressions and of the maternal nutrition upon the development of the embryo; and the part, if any, played by the nervous system in the production of abnormalities during the same period.

Section III.—Consideration of certain possible causes of Malformations.

Part 1.—*Mental Impressions.*

Mental impressions of the pregnant woman reacting upon the developing fœtus were for years supposed to be a potent factor in the production of abnormalities. This question is an important one, since abnormalities so produced would be undoubtedly somatogenic. The theory has, however, been long abandoned by, I believe, all biologists. Vrolik * years ago formulated his reasons for denying its action. These reasons are to my mind unanswerable, and, in order that this paper may be as complete as possible, I shall reproduce them here with some additional comments.

(1) As Allen Thomson points out†, "It may be remarked that the stage of the period of pregnancy at which the injury of the child may take place is by no means defined, and that there is no correspondence between the time or advancement of the fœtus and the nature of the injury. Some injuries are said to have occurred or to have had their foundation laid at the very moment of conception, and even occasionally before that time, while others are inflicted only a few weeks before birth." Where by chance the time of the supposed "impression" does coincide with the malformation, and this is the case in a very small minority of instances, there is no reason to suppose that the occurrence is other than a coincidence, for the reasons which follow.

(2) "That malformations seldom, or perhaps never, agree with apprehensions or fears *à priori* of pregnant women (G. Vrolik, T. Zimmer, J. J. Plenck, and Burdach). On the contrary, it often happens that a woman who has once procreated a malformation, and is continually troubled by the fear of another similar sad occurrence, may become the happy mother of a second well-formed child." (*Vrolik.*)

(3) There is no nervous connection between mother and child through which such an impression could act. There is abundant evidence to show that a violent mental shock to the mother may cause the death of the fœtus and its subsequent expulsion. Thus Priestley ‡ says :—"There is no doubt greatly increased nervous

* Todd's Cyclop. of Anat. & Phys., Art. "Teratology."

† *Ib.* Art. "Generation." ‡ Pathology of Intra-Uterine Death, p. 68.

tension in all pregnant women. Fright, anxiety, a sudden impression made upon the mind or body, may not only initiate uterine contraction at any period of pregnancy, but there is every reason to believe that a sudden mental shock may at once kill the early embryo or more mature child even if it be retained some time afterwards. Repeatedly it has occurred to me, as to others in practice, to have patients dating the exact time of their child's death *in utero* to some alarm of shocking occurrence which has profoundly affected the whole nervous system. The immediate effect described was first violent perturbation and undue active movement of the child for a brief interval, followed by cessation of fœtal movement, and absolute quiescence in the future. In a few rare instances the woman has been mistaken in supposing her child to be dead after a shock or fright she has experienced, but in a large number of cases the relation between the cause and the effect has been too clear to be accounted for by mere coincidence, and the child has sooner or later been expelled dead, possibly both dead and putrid. It is no uncommon thing for a woman in early pregnancy, on the receipt of bad news, which much perturbs her, to be seized with uterine hæmorrhage, ending in abortion. Condemned women prior to execution have been known to abort beforehand, and, under the influence of terror and pain, martyred women in former days are said to have aborted at the stake. So potent is mental influence on the stability of pregnancy, that I have had reason to believe the mere dread of miscarriage has in some women been an important factor in bringing it about; and I have known pregnant women who have previously miscarried, get into such violent mental agitation as the time approached at which they had aborted before, that the event they feared was precipitated, and pregnancy was brought to a premature conclusion."

It is not difficult to understand how, by undue oxygenation of the blood leading to fœtal asphyxia, or by some chemical alteration in its constitution, at present unrecognized, the death of the embryo might be caused, but it does not appear how its mal-development can be accounted for by similar causes.

(4) Malformations, and those of a similar nature to those met with among human beings, occur amongst lower animals, where the effect of an "impression" can scarcely be postulated.

(5) One of a pair of twins may be malformed, whilst the other escapes.

(6) "More deeply situated organs, the very existence of which may be unknown to the pregnant woman, may be malformed; as, for instance, the heart, the intestinal tube, &c." (*Vrolik.*)

Finally, I may conclude this section with the words of Thomson:—"We conclude by adopting and expressing the words of Dr. Blundell, 'that it is contrary to reason, experience, and anatomy to believe that the strong attention of the mother's mind to a determinate object or event can cause a determinate or a specific impression upon the body of her child without any force or violence from without; and that it is equally improbable that, when the imagination is operating, the application of the mother's hand to any part of her own body will cause a disfiguration or specific impression on a corresponding part of the body of the child.'"

Part 2.—*Effect of Maternal Nutrition.*

The contention may possibly be raised, as has been hinted in earlier sections, that the state of the mother's nutrition during the period of pregnancy may be a factor in the production of abnormalities. The forms in which this kind of influence might *à priori* be expected most probably to take effect are those of dwarfs and giants, but as I endeavoured to show, when dealing with those groups, there is no evidence of this factor being of any importance; indeed there seems, on the contrary, good reason for the formation of an opposite opinion. The case of the lion-cubs affected with cleft-palate is, however, one which at first sight seems to lend some colour to such an hypothesis. In considering it, however, it must not be forgotten that these were not hereditary cases, but had, in all probability, a specific cause, and ceased to occur when that cause was removed. On the other hand, there are numbers of cases which might be cited where faults of excess and of defect co-existed in the same children, to explain both of which by the nutrition hypothesis would seem to be a very difficult matter. Thus, for example, in the Heinzler family polydactylism and defective development of the teeth co-existed. And, again, and this case is of special interest in connection with the lion-cub matter, Roux * has recorded an instance where a father and child were both the

* Lucas, *op. cit.* vol i. p. 307.

subjects of hare-lip and cleft-palate, and both also possessors of six digits. I may again revert to the views of Geddes and Thomson on the subject of the influence of nutrition on the determination of sex. Should it be the case that this is the determining factor, it would be a strong argument in favour of a direct somatic influence upon the germ-plasm, though even then it would not prove that the state of the maternal nutrition had anything to do with the production of abnormalities. Meantime the hypothesis just referred to requires a great deal more proof before its acceptance can become at all general.

Medical literature is full of cases illustrating the influence of the state of health of the parents, both male and female, in producing the early death of the fœtus, or the birth of sickly, ill-developed children. Dr. Priestley states that he knew of one case where a man the subject of slight albuminuria married a young woman apparently in perfect health. They had one child, delicate and fragile, within a year, and the wife aborted subsequently in three successive pregnancies—the husband growing weaker year by year, and eventually dying of uræmia. Lead-poisoning and other affections of the parents, most notably of all syphilis, may be followed by the same results. Again, as Stolz * has observed, fat women are often sterile and if they conceive are apt to abort. He believes this depends on nutrition taking an abnormal direction, and that the nutritive fluids destined for the nutrition of the embryo are thus insufficient for its development. All these influences, however, produce, as might be expected, general effects upon the whole fœtus, and not isolated or scattered abnormalities, at least so far as we at present know.

Part 3.—*Effect of Placental Diseases.*

Various diseases are known to attack the placenta, in many cases causing the death of the fœtus. Is it possible that these may in any instances lead to the production of abnormalities where insufficient to cause the death of the fœtus? I have searched through a considerable amount of medical literature without much result. Ercolani † does not mention any such possibility. In

* Des Accouchements (quoted by Priestley).

† Histology and Pathology of Reproduction.

fact the only note on the subject which I have been able to find is a remark by Priestley that Spath in nineteen cases in which calcareous concretions were present in the placenta, found one of congenital rachitis, one of spina bifida with hydrocephalus, and one of slight hydrocephalus. On which Priestley remarks that "the presence of calcareous deposits, in the placenta, therefore seems associated with some pathological conditions unfavourable to the welfare of the child, although the adverse influence does not proceed far enough in most cases to extinguish life." It is to be noted that the first and last of the cases mentioned are cases of fœtal disease, and that the second one, seeing that hydrocephalus was also present, almost certainly comes under the same category. As in the case of parental nutrition, it seems more likely that disease of the placenta would produce general rather than special effects upon the fœtus.

Section IV.—On the possible Nervous Origin of certain Malformations.

Part 1.—*Affections of a possibly Trophic Nature.*

Under this heading I desire to discuss the possibility of an inefficient action of the nerves during the development of the embryo being a cause of malformations. I have used the word "trophic," not because it quite expresses what I mean, but because I know of no better term. The point I wish to raise is, whether the trophic nerves or other nerves allied to them have not a potent action in directing the development of the embryo, and whether a failure for any reason on the part of these nerves to do their work may not be followed by certain malformations. In considering this question, it will first be advisable to learn what effects follow upon trophic failure in post-uterine life. Landois and Stirling * say in this connection, that the nutritive changes which follow in the eye, upon section of the ophthalmic division of the fifth nerve, are best explained by the theory of trophic fibres, whose centre is the Gasserian ganglion, and they proceed to state :—"The trophic disturbances which sometimes accompany affections of the trigeminus are particularly interesting. They

* Text-Book of Human Physiology, vol. ii. p. 796.

are—a brittle character of the hair, which frequently becomes grey or falls out; circumscribed areas of inflammation of the skin, and the appearance of a vesicular eruption upon the face (often following the distribution of certain nerves), which may also occur on the cornea, constituting the neuralgic herpes corneæ of Schmidt-Rimpler. Lastly, there is the progressive atrophy of the face, which is usually confined to one side, but may occur on both sides. It is caused very probably by atrophic affection of the trigeminus, although the vaso-motor nerves may also be affected reflexly." The following cases will illustrate the effects:—

(1) Otto Schmidt* was first affected at the age of ten, atrophy of the left side of the face commencing at that age. At the age of forty-one, the muscles, bones &c. of the left side of the face were much smaller than those of the right, and all the subcutaneous fat had quite disappeared, no hair save a very small moustache growing upon that side. The left orbit was much larger and the eye deeply sunken from disappearance of the post-orbital fat. Atrophy did not extend beyond the vertex. The median line of the face was crescentic, with the concavity directed to the left, from the shrinking of that side. The left side of the tongue was atrophied, and the sight of the left eye impaired, though not from atrophy of the optic nerve. The sense of smell and discharge of mucus were both less on the left side than on the right. All these changes were probably due to some lesion of the trophic fibres of the trigeminus.

(2) Dr. Stewart† has recorded a case where the patient, a boy, aged 14, was severely frost-bitten at the age of ten, on the left cheek and ear. Eighteen months after, atrophy was first noticed. Two years later, when he came under notice, atrophy affected those parts of the face innervated by the two lower divisions of the fifth nerve. The skin, subcutaneous tissue, muscles, and bones were all atrophied, the muscles least. The lower jaw was thinner and shorter, and the upper distinctly atrophied on the left side. The teeth were well developed. Owing to atrophy of the turbinated bones, the left nostril was wider than the right. There was distinct atrophy of the left half

* The account is taken from a note made when Prof. Purser showed the patient to his class in the University of Dublin.

† Montreal Med. Journ.

of the tongue, more marked anteriorly. There was no affection of any of the special senses, nor any disturbance of deep or superficial sensation, nor was there any difference of temperature between the two sides.

(3) Mendel* has reported the results of a very thorough examination he made of the fifth nerve in a case of facial hemiatrophy, of many years' standing, in a woman. This woman had also atrophy in the region innervated by the left musculo-spiral nerve. She died from phthisis. Her case was first described by Romberg and more recently by Virchow. The symptoms were those of a typical left facial hemiatrophy. Mendel found all the branches of the left fifth nerve, from their origin to their termination, the seat of a proliferating neuritis. A marked and similar difference was found in the size of the right and left descending roots of the fifth nerve, and also in the substantia ferruginea, the alleged nucleus of the so-called trophic root of the fifth nerve. This examination shows that, at least in some cases of facial hemiatrophy, we have to do with a neuritis of the fifth nerve.

Such being the effects of trophic lesions in post-uterine life, we have now to consider what evidence there is for any similar affections occurring in the developing fœtus. We know, unfortunately, so very little about the directing causes of development in the embryo, that all speculation of this nature must be somewhat hazardous. I shall now detail such cases as I have met with, as seem to lend probability to such an action of the nerves during development as I am arguing for.

(1) I have first to revert to the exceedingly interesting remarks made by Oakley Coles, in his chapter on the etiology of cleft-palate, to which I have already referred, but which must now be more particularly considered. "The frequent association," he says, "of cleft-palate with defective development of the brain has long been observed †, and various hypotheses have been put forward to explain the connection. Thus, in the early part of the century, Tiedemann ‡ observed that in certain cases of cleft-palate the nerves of smell are wanting or imperfectly formed, and

* Neurol. Centralbl., July 15, 1888.

† Leuckart, 'Untersuch. üb. den Zwischen Kiefer-Knochen des Menschen,' Stuttgart, 1840.

‡ Zeitschr. f. Phys. Bd. i. S. 71.

he was therefore inclined to attribute the deformity in the palate to a deficient development of the framework of the olfactory organ consequent upon the nervous defect. This view, however, never met with any general acceptance, and M. J. Weber (no mean authority on the subject) states * that he has never seen the olfactory nerves absent in any case of fissured palate. According to Dr. Engel, on the other hand, the deformity is due to increased breadth of the anterior portion of the head, caused by a variety of conditions of embryonic life, such as hernia cerebri, dropsy of the third ventricle or of the lateral cornua of the lateral ventricles, or excessive development of the anterior cerebral lobes. Or, in other words, to a purely mechanical disturbance of the relative position of the parts involved. But while admitting the accuracy of Dr. Engel's observations, it is impossible to accept his deductions from them. For cleft-palate is found to occur more frequently in connection with a microcephalic skull than under the conditions quoted by Dr. Engel; and thus his hypothesis, even if true, would only explain the causation of a limited proportion of cases. It is far more probable, however, that the relations between the two deformities, the cerebral and the palatal, is not one of causation, but one of concurrence, both being common effects of a grave vice in the developmental energy of the fœtus. . . . All, perhaps, that we can safely say on the subject at present is that cleft-palate, hare-lip, and other similar anomalies of development do frequently occur in conjunction with faulty development of the brain, whether bilateral or unilateral, hypertrophic or atrophic; and it is quite possible that the two kinds of deformity may be related to each other as cause and effect. But the facts that hemicephalic and microcephalic infants are born with perfect palates, while the subjects of palatal deformity are in many cases of high intellectual power, would appear to show that the two deformities are rather the combined effects of a common cause." Again, after speaking of the perfect condition of the palates of ancient and modern uncivilized races, and comparing their state with that of civilized races, he says :—" We shall be led to the inevitable conclusion that the relation between a high state of civilization and a high proportion of palatal deformity is something more than a mere matter of coincidence; and the strength of such conclusion will not be lessened by the state-

* Froriep's Notizen, Bd. xix. No. 18, S. 282.

ments of Walther* and Langenbeck †, both of whom maintain that the severer forms of cleft-palate have become more common within their own recollection. To draw more precise conclusions from considerations such as the above," he proceeds, "would be beyond the scope of the present article, but it may not perhaps be out of place to suggest that the difference between the conditions of civilized and uncivilized life is quite as much a matter of increased nervous strain as of changed physical environment; that the over-taxed nervous system, which in the parent manifested itself only by functional instability and subjective remonstrance, may, in the child, issue in objective defect and an actual refusal to complete its alloted task."

(2) Dr. Langdon Down has drawn attention to the occurrence of palatine abnormalities in congenital idiots. Out of two hundred cases observed eighty-two "possessed palates inordinately arched, and with this increased arching were noticed various abnormalities. In seven the palate-bones did not meet, leaving a sulcus between them, the mucous membrane being, however, continuous. There was no instance of the ordinary cleft-palate, and I may remark that in an examination of nearly six hundred idiots, I have failed in meeting with an example of that deformity. In several the hard palate extended but a short distance posteriorly from defect of the palatal process of the superior maxillary bone and entire absence of the palatal bone, and in all these cases the velum palati was unusually flaccid. In the majority of cases there was marked narrowness of the palate" ‡.

(3) In describing cases of hereditary ataxia, or Friedreich's disease, my colleague Dr. Suckling says that the association of the disease with other deformities is interesting. "In the one family, one son was born with deformity of the foot, and a daughter with only one upper limb. In the other family two cousins were born bald. There is no doubt that the deficiency in the nervous tracts is a congenital one, and due to a fault in development" §.

(4) Beigel ‖, in a paper on albinism and nigrism, gives it as his opinion that these conditions are due to nervous affections.

* Graefe u. Walther's Journal, Bd. xxi. S. 175.

† Neue Bibliothek für die Chirurgie, Bd. iv. Hft. 3, S. 492.

‡ Mental Affections of Childhood and Youth, p. 159.

§ Repr. from Illustrated Med. News, 1890.

‖ Virch. Arch. xliii. 529.

(5) Stricker * gives the case of a family where, amongst hair otherwise perfectly black, a white lock existed. This abnormality ran five generations, the first individual known to possess it having lived in 1720, and the case having been described in 1877. If Beigel's view be correct, this may also have been due to a nervous defect. It is, indeed, difficult to know how else to account for it.

(6) Mr. Lloyd Owen † has described carefully a case where congenital nystagmus was transmitted through four generations. This condition appears to be due to some congenital defect of the nerve-centres.

(7) Bland Sutton ‡ gives the following case.—"A woman in the fifth month of gestation fell downstairs on her abdomen. At the eighth month she was delivered of a child, the upper part of the body presenting the proportions of a fœtus of corresponding date, but all parts below the navel agreed with those of an embryo of the fifth month of intra-uterine life. Dissection showed that the spinal column ended at the first lumbar vertebra, the remaining lumbar, sacral, and coccygeal elements being absent. The skin of the legs was exceedingly thin, and, on reflecting it, the bones were found to be thin and to present the characters of those of an embryo of the fifth month. All the other tissues of the legs, muscles, nerves, ligaments, &c., were represented by adipose tissue. In this instance it is probable that when the mother fell, she fractured the spine of the fœtus; the result was to cut off nervous influences from the legs, which in consequence retrograded into fat." He refers to other examples of this fatty degeneration subsequent to loss of nerve-influence §, and says that "there seems to be, as Otto ‖ was the first to demonstrate, some intimate relation between absence of nerves and fatty degeneration; and he points out that parasitic fœtuses, which, as a rule, are devoid of nerves, always contain a very large quantity of fat in lieu of more important tissue-muscles and the like."

(8) Furst ¶ narrates a case where chronic hydrocephalus was accompanied by cessation of growth.

* "Noch eine Familie von Haarmenschen," Virch. Arch. lxxiii. 622.

† Ophthal. Rev. vol. i. p. 239.

‡ Introduction to General Pathology,' p. 85.

§ Med.-Chir. Soc. Trans. lxviii. p. 293.

‖ Compendium of Human and Comp. Path. Anat. (South's transl., 1831).

¶ Virch. Arch. xcvi. 357.

(9) Gowers * has described the condition of the brain in a case of congenital absence of one hand. The subject was a male, aged 40, who was born without a left hand. The forearm bones were well developed, but at the extremity there was only an irregular mass of bone consisting apparently of the two rows of carpal bones, very imperfectly developed and anchylosed together except at one point. In the brain there was a marked difference between the two ascending parietal convolutions. At their origin at the longitudinal fissure, for the first inch of their extent, they were nearly equal in size, and continued nearly equal for the upper $1\frac{1}{2}$ inches. In the next (middle) two inches there was a very marked difference, the right being a narrow single convolution, and the left broad and depressed by a slight secondary sulcus. This occupies precisely the area, stimulation of which, according to the experiments of Ferrier upon monkeys, causes movements of the opposite hand. It is, of course, impossible to say whether the brain or the hand defect was the primary one. The following remark of the author, however, renders it possible that the former may have been the cause:—"I am not aware that the brain has been examined in any similar case. In several instances, in cases of old amputations of the arm, an atrophy has been found, but it has been slight, and has not been uniformly localised."

These facts, I think, render it probable that the nervous system exercises more influence upon the course of development than has been hitherto attributed to it. It is true that, on the opposite side, it may be urged that anencephalous fœtuses are fairly well developed and are yet without brain. To this it may be replied—1st, that the development is more apparent than real, since the bodies of such fœtuses are always overloaded with fat, a condition which, as we have seen, follows upon loss of nerve influence; and 2nd, that we do not know the date at which the disease occurs which causes the defect. Certainly it is later than the period at which the eyes are fully formed. It may be that the disease does not occur until after development has proceeded sufficiently far to proceed with the remnants of nerve system which exist. It must not be forgotten that these forms sometimes live and breathe for a short time, showing the existence of some important parts of the nervous system.

As regards the kinds of malformations most likely to follow

* 'Brain,' Oct. 1878.

upon a loss of nervous influence, it appears to me that many of the clefts can best be accounted for in this way. For example, it is a little difficult to see how a branchial fistula can depend upon a lack of material, since the aperture at the time of failure to close must be excessively small. On the other hand, it is not difficult to see how a failure of power to close, due to a want of nerve influence, may cause the persistence of the cleft. A similar explanation may be offered for some of the forms of cleft-palate where there is no apparent lack of material. The co-existence of several malformations in the same region seems to point to some common cause, which is most probably to be sought for in the nervous system. Such grouped malformations occur most often in the face in connection with ears, eyes, and palate. It is suggestive to observe that these are in the region of supply of the trigeminus nerve, and that, as Anstie * observes, "the nervous centre in which the trigeminus is implanted is, of all nervous centres, the one which in the human subject is most liable to congenital imperfection of the kind which necessitates a break-down in its governing functions at special crises in the development of the organism."

Here at present I must leave the subject of the connection of nerve influence and congenital lesions; at a later part of this paper I shall recur to it in relation to the origin and development of a malformation.

Part 2.—*Affections due to Excessive and Irregular Nerve-impulse and Muscular Contractions.*

It is, of course, a well-known fact that the child after a certain period of intra-uterine existence is capable of making a considerable amount of use of its muscles, and, moreover, that the amount of movement varies in different children. Many have held that an excess of this movement, exercised with irregularity, is the cause of various malformations. Talipes is that which has been most commonly attributed to its influence; thus Lowne † groups the various forms of that defect under the heading of "Distortion from irregular muscular contraction." These malformations, he says, "are probably due to some form of cerebro-spinal irritation or defect." Again, speaking especially of the various forms

* Lancet, 1866, i. p. 654.

† Teratological Catalogue of Roy. Coll. of Surgeons of England.

of talipes, he says, "The question of their origin from irregular muscular contraction is still an open one; but the arguments in favour of this view are exceedingly strong." Messrs. Parker and Shattock, as has before been mentioned, take a different view as to the etiology of the condition. In their specimen it is true that a microscopical examination revealed no lesion in the central nervous system; at the same time it is a matter for argument whether this is proof positive that no nervous influence was concerned in the production of the condition. It seems to me at least possible that some temporary stimulus, say of a chemical nature, might set up irregular contractions in the muscles, without any changes resulting in the nervous system. We know that drugs administered to the mother can affect the fœtus, and the same may be true of other chemical stimuli of which we may know little or nothing. If this be true, the resulting lesions would be somatogenic in their nature; but we are at present in ignorance as to whether, in the first place, all lesions which are attributed to this cause are really due to it, or whether some of them may not follow upon a lack of material, as seems probable, in which case they would be blastogenic in their nature. And, secondly, supposing that these lesions owe their origin to more than one cause, we have no method, at present, of distinguishing between the two or more classes. Here, as in many other instances, it is much to be hoped that teratological workers will direct, in the future, more attention to the causation of malformations, so that these and other cognate problems may be cleared up.

Section V.—Hereditary Disease.

The subject of hereditary disease is one which should not be neglected in an inquiry of this kind. I purpose, therefore, devoting a few lines to this subject in its connection with the present topic.

The greatest confusion has been introduced into the controversy on acquired characters by some who have mixed up the various kinds of hereditary diseases, which fall into classes of the most divergent nature, with one another and with hereditary variations and malformations. It will, then, be advisable to state what are the groups of hereditary diseases considered in relation to the present inquiry.

(1) There are diseases due to a specific infection, probably always bacterial in its nature. Small-pox and other similar diseases, with which the fœtus may be infected by the mother, are of this kind. The commonest and best example is, however, that of syphilis, which may be communicated to the embryo by its mother, or to the embryo by the father, and by the embryo, in turn, to its previously uninfected mother. Diseases of this kind have no bearing whatsoever upon the present question, though it is sometimes imagined that they have.

(2) There are conditions of the embryo induced by the presence of a poison, not bacterial in its nature, which may be present in the parental organism at the time of impregnation. To this group may possibly be assigned the cases of early intra-uterine death or congenital feebleness of the embryo, which, as has been already stated, are caused by lead-poisoning in the male parent. Those cases, also, where feebleness of mind or body in the child seems to be the result of chronic alcoholism in the parent or parents. In connection with this subject I cannot refrain from mentioning the remarkable statements of Dr. Langdon Down * as to the effects of intoxication in the parents on the offspring. The case which he gives is that of a child (female) aged five years, without any deformity, but only 22 inches in height, and unable to speak. The first child of the family was healthy. Prior to the procreation of the second the father took to drink, the offspring dying at the age of three years, and during its life resembling that described above. The third was the child first mentioned; and the father was drunk when he procreated it. The fourth was a miscarriage. At this period the father became again a sober man, and his wife had subsequently five perfectly normal children. The above case, the author says, is of great interest, because it adds another to a group of cases which have come under his observation, of arrested development arising from the intoxication of one or both of the progenitors at the time of the procreative act. The whole group of cases has presented features of such close resemblance that it is difficult to avoid the conclusion that there was some unity of cause, and careful investigation has elicited facts bearing on the etiology of these cases having a close parallelism to the circumstances which he believes to have been potential in this. He has known some of these

* Trans. Path. Soc. xx. 419.

cases to attain the age of twenty, while still preserving infantile characteristics. If these deductions be true, it certainly would seem as if the soma was capable of exercising a greater influence upon the germ-plasm and its development than some writers are prepared to admit.

(3) There are the diseases commonly called hereditary, such as gout, rheumatism, phthisis, and hæmophilia. These require a little consideration. The first two are due to some fault in the chemical processes in the body, and at first sight seem to have little to do with the subject of congenital malformations. I believe, however, that this is more apparent than real, and that, as a matter of fact, they are more closely allied than on the surface appears to be the case. It is true that it is the chemical process which is at fault, but the primary flaw must surely be in the laboratory in which they take place. To say that we do not know the physical explanation of the failure, is no more than to say that we have not yet penetrated all the mysteries of human pathology. In the hereditary cases it would seem that the parent transmits to the offspring a flaw or weakness in the chemical apparatus, which under strain leads to its subsequent breakdown and the appearance of the characteristic disease. Such a flaw or weakness may surely with correctness be called an hereditary malformation. Again, as regards phthisis, the tubercular bacillus is only one factor in the disease: there must also be a suitable nidus or soil for it to take root in; the condition of tissue, whether intestinal, pulmonary, or otherwise, which provides this suitable soil is often hereditary, and may fairly be looked upon as an hereditary malformation. As regards hæmophilia, there can be little difficulty in seeing that the hereditary malformation consists in some faulty construction in the walls of the blood-vessels, which prevents them from behaving in the normal manner when cut or torn. As regards cancer and its heredity, since we are quite ignorant as to the cause of that complaint, it is useless trying to form any theory to account for its transmission. In the next and concluding section, I shall have to discuss the bearing of these facts upon the question involved in this paper.

Section VI.—Conclusion.

I have at various points in this paper had to allude to the unfortunate gaps in our teratological knowledge, which make the drawing of indisputable conclusions so very hazardous. I shall therefore content myself with briefly indicating those points which have chiefly struck me in working at the subject. Even if the deductions be inaccurate, the facts and words of others collected in this paper may render it of some service to others working at the same subject, and probably especially so to those who are not members of the medical profession, and who are therefore perhaps less conversant with its literature than those who, like myself, are in the constant habit of referring to it. I will now mention the points to which I have above alluded.

(1) It is an interesting point that those malformations whose blastogenic nature is least in doubt are, speaking generally, those also whose hereditary nature is most distinct. I would refer, as an example, to polydactyly.

(2) Again, it is interesting that those malformations which are undoubtedly somatogenic are, so far as I know, non-hereditary. I allude to the abnormalities described in Section II. Part 3, but the remark just made must be taken with the limitation that so many of these forms are still-born or survive but a brief period. It might be thought that a further limitation should be made on account of the difficulty that gravely deformed persons might find in getting married; but this is, I think, an unnecessary limitation. The study of teratological literature almost seems to teach one that any person of either sex can get married if they desire it. Let me give an example from both sexes. Butcher * has figured a woman and her child both affected with the most aggravated form of double hare-lip and cleft-palate, than which scarcely anything can lend a more horrible appearance to the face. Butcher operated upon both at the same time and remedied the defects to a large extent. On the other side, I knew of a man quite destitute of both upper and lower extremities, who was not only married but the progenitor of a well-formed and handsome progeny. I do not know the cause of the defect in his case.

(3) It must be admitted that besides the cases which have been alluded to in the above two sections, there remain still a number of others as to which no definite conclusion can be, at present,

* Dubl. Journ. of Med. Sci, lxiii. 426.

arrived at. These have been already sufficiently indicated in the earlier sections of this paper, and need not, therefore, be now further specified. Much more extended observations will be required in most of these cases before it will be possible to settle the question as to their nature.

(4) There are certain malformations which suggest the possibility that they may have been gradually acquired and subsequently transmitted to descendants. Some of these may now be briefly mentioned. The question of hereditary myopia and hypermetropia is one which Weismann has considered in his essay "On Heredity"*. He there states:—"Those fluctuations on either side of the average which we call hypermetropia and myopia, occur in the same manner, and are due to the same causes, as those which operate in producing degeneration in the eyes of cave-dwelling animals. If, therefore, we not unfrequently meet with families in which myopia is hereditary, such results may be attributed to the transmission of an accidental disposition on the part of the germ, instead of to the transmission of acquired short-sightedness. A very large proportion of short-sighted people do not owe their affliction to inheritance at all, but have acquired it for themselves; for there is no doubt that a normal eye may be rendered myopic in the course of a lifetime by continually looking at objects from a very short distance, even when no hereditary predisposition towards the disease can be shown to exist. Such a change would of course appear more readily if there was also a corresponding predisposition on the part of the eye. But I should not explain this widely-spread predisposition towards myopia as due to the transmission of acquired short-sightedness, but to the greater variability of the eye, which necessarily results from the cessation of the controlling influence of natural selection." I have already mentioned that Mr. Priestley Smith has stated that there is a supposed correlation between the growth of the brain and the growth of the eye, by reason of which a high degree of cerebral development is apt to be associated with an over-development of the eye. If this be so, apparently the brain condition is the primary factor, at least in a certain number of cases. But the brain condition may be due to variations in the germ itself and, therefore, blastogenic in nature, and the inheritance of the defect might follow without any necessity for an appeal to the heredity

* English ed. (Poulton), p. 89.

of an acquired defect to account for it. But it is a question whether an explanation such as this can be held to account for all the cases of hereditary myopia. In his essay, however, "On the Supposed Botanical Proofs of the Transmission of Acquired Characters" *, the author seems to admit a modification of his views as first expressed, which are of great importance in connection with the question of the inheritance of the group of defects at present under consideration. Dr. Mivart in reviewing these essays † makes the following comment on this modification:—"Although these last two essays are intended to show that acquired characters cannot be inherited, they yet seem to us to show that to a certain extent, and in a certain sense, they may be inherited. We have no desire to contend that they are heritable to any large extent, and we have always affirmed that mutilations can at the most be very rarely inherited, and long ago referred to obvious proofs that so it must be ‡. But Professor Weismann here certainly makes some admissions with respect to the cumulative effect of a changed environment on the germ-plasm of organisms, which contradict his previous assertions that only unicellular creatures can be thus modified. But if such a cumulative effect does exist, then, if sufficient time be allowed (and Darwinians are prodigal of time), a modified Lamarckism reappears!" It is possible that in the direction thus indicated an explanation may yet be found for some of these conditions. But the whole subject, so far as the eye-defects are concerned, wants working out thoroughly by practical ophthalmologists.

(5) There are certain points which should be noted, since they seem to indicate the gradual rise and development of a malformation. I will mention two examples. The first is that of the precursory conditions, so to speak, of cleft-palate and hare-lip which were mentioned when that subject was under consideration; these are of great interest. Again, in connection with the same subject, it should be mentioned that Lucas § has published observations which lead him to conclude that an absence of incisors in a parent is premonitory of cleft-palate or hare-lip in the children, and he has given several cases in support of this theory. I am bound to say that my colleague Mr. Humphreys and myself, when working

* *Ib.* p. 413. † Dub. Review, 1889, p. 269.

‡ Genesis of Species, 2nd ed. p. 242.

§ Brit. Med. Journ., Dec. 3, 1887, p. 1212.

at the subjects of increase and diminution of the incisors *, did not meet with any cases which bore out this theory; but this, of course, does not prove that in certain cases it may not be true. If so, it strengthens the argument in favour of precursory conditions for some cases of palatal and labial defect. The second example is quoted by Page † from Boehm ‡. A woman had "beautiful blue eyes, delicate white skin, and, what is especially characteristic of a tendency to albinism, colourless eyebrows and eyelashes." Her daughter had white eyebrows, lashes, and skin, and great irritability to light, whilst the grand-daughter had internal strabismus and nystagmus, and hair "originally as white as well-bleached linen." Some other cases of a cumulative nature are given in my previous paper. It seems to me possible that these cases are due to an original flaw in the nervous system. Whether this flaw is due to a failure of development consequent upon conditions inherent in the germ, or upon the slow effect of some condition connected with the environment acting upon several generations, as was suggested by Coles when writing about cleft-palate, and as Weismann seems to hint may be the case in the essay referred to above, must at present and for a long time remain an open question. But in either case the nervous defect would precede the more obvious one, and may in an earlier generation exhibit its effects in a manner perhaps never recognized, by slight trophic disturbances and the like. Descending further and gathering force as it descends, under favourable circumstances, the conditions met with, at times, in the parents of children with cleft-palate or whatever the defect may be will be reached, and in the next generation or in one closely succeeding the full defect may appear, the precursory conditions having been quite unnoticed. I do not wish it to be understood that I am arguing either that all malformations have a nervous origin or that the chain of events which I have suggested, or one of a similar nature, occurs in all cases; what I desire to point out is that in some cases, and perhaps in more than at present we have any idea of, such precursory conditions may be capable, by diligent inquiry, of demonstration. In these facts we also may, I think, find a clue to the real significance of the much-abused word "tendency."

This word, and especially in its relation to the so-called here-

* Journ. of Anat. and Phys. vol. xxi. p. 84. † 'Lancet,' Aug. 8, 1874.

‡ Der Nystagmus und dessen Heilung.

ditary diseases, means that there is at present in the subject a minor condition of the nature of a congenital malformation. It is possible that under the most favourable conditions of life for the given defect it may never lead to a breakdown or become apparent, whilst under other and less favourable circumstances the weak point may give way and the defect become obvious. Such an explanation as this might be without hesitation accepted as accounting for the varieties of eye-defects recently referred to; and I believe that further investigation will show, as far as demonstration is possible, that the same line of argument may be used, not only with regard to the remaining diseases of the hereditary group described as (3) in Section V., but also to some at least of the congenital and hereditary malformations as well.

A Revision of the *Forficulidæ*, with Descriptions of New Species in the British Museum.

By W. F. Kirby, F.L.S., F.E.S., of the British Museum (Natural History).

[Read 19th June, 1890.]

(Plate XII.)

The Forficulidæ, or Earwigs, have hitherto attracted less attention from Entomologists than any other group of Orthoptera. There are but few papers of any importance on the group; those by Dohrn in the 'Stettiner entomologische Zeitung' (vols. 24–26, 1863–1865), which include a descriptive synopsis, and Scudder's Catalogue of all the described species, in vol. 18 of the 'Proceedings of the Boston Society of Natural History' (1877), being the most useful. Since then several species have been described by De Bormans, Karsch, and others in various foreign periodicals.

Having lately rearranged this group in the British Museum, I judged that it would give a useful impetus to its study to publish a revised synopsis of the genera on the lines already indicated by Dohrn and De Bormans, and to describe as many new species as were before me in sufficiently good condition. Several of these were ticketed with MS. names by Prof. Westwood, which I have usually adopted.

One great drawback in the study of the Forficulidæ is deficiency of material. They are usually received in very small numbers and rarely in quite perfect condition, the antennæ, which are